11 Bioaccumulation in the assessment of sediment quality: uncertainty and potential application

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The desire for cost-effective screening tools for contaminated sediments has resulted in the development of a variety of numerical sediment quality guidelines (SQGs). Currently developed guideline values can be categorized as either mechanistically based (e.g., equilibrium partitioning [EqP]) or empirically based (threshold effects levels [TELs], probably effects levels [PELs], effects range low [ERLs], effects range median [ERMs], apparent effects thresholds [AETs]) (Chapters 3 and 4). None of the existing approaches in either of these categories were designed or intended to be protective of indirect effects through bioaccumularion Thus, there is a need (and under certain United States [US] regulatory programs, a requirement, e.g., the Marine Protection, Research and Sanctuaries Act [MPRSA] and the Clean Water Act [CWA]) to assess the potential for sediment-associated contaminants to bioaccumulate and to evaluate any porential effects (direct and indirect [i.e., foodchain]) associated with that bioaccumulation. While the technical basis, utility, and accuracy of existing SQG approaches in predicting direct effects to benthic infaunal organisms is discussed in other chapters (e.g., 4, 12, and 13), the intent of this chapter is to provide an overview of the potential for guideline values to predict effects through bioaccumulation.

Bioavailability and organism physiology are the two most important variables affecting chemical contaminant body burdens (Landrum et al. 1996). Organic carbon (both content and composition), contact time (aging), source (e.g., polycyclic aromatic hydrocarbons [PAHs] that are petrogenic in origin versus byproducts of combustion), and sediment surface area, howev-

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er, can also affect the proportion of nonionic hydrophobic organic compounds available for uptake by organisms. For organic compounds, the octanol–water partition coefficient (K_{ow}) may be the single best predictor of partitioning, but it is insufficient for completely determining contaminant behavior and its bioavailability in the environment. For compounds with ionizable groups, the pH of the environment also affects contaminant bioavailability. Thus, 2 factors, organism lipid content and sediment organic carbon, control to a large extent the partitioning behavior of organic compounds between sediment, water, and tissue. The more hydrophobic a compound, the more likely it is to associate with nonpolar (nonionic) matrices (lipid and organic carbon).

Metals and ionizable organics, on the other hand, are more complicated because their bioavailability can be controlled by a multitude of inorganic and organic ligands, such as iron and manganese oxyhydroxides, acid volatile sulfides (AVS), organic carbon, and others (Chapters 4 and 13). The degree to which some of these factors control metal bioavailability can vary greatly as a result of changes in redox state and hydrogen ion activity (Tessier and Turner 1995). One active area of research is focused on the relationship between AVS in sediment and concentrations of metals in water that are presumably available for uptake (e.g., Di Toro et al. 1990; Ankley et al. 1993; Hare et al. 2001; Sundlin and Eriksson 2001; Chapter 13).

While all organisms bioaccumulate (primarily organics), there are often large differences in tissue residue concentrations between species exposed to the same sediment. Similarly, there can be large differences for the same species exposed to different sediments or the same sediment at different times. The tissue residue concentration of a contaminant is a function of the environmental concentration (including route and source of exposure), duration of exposure, and a given species' ability to metabolize accumulating compounds. Organismal factors, such as cellular lipid level and rate of uptake and elimination (metabolism, diffusion, and excretion), are the primary determinants of tissue residue concentration and time to reach steady state. Often, the highest concentrations can be found in internal organs, such as the hepatopancreas, because of the high lipid content. In addition, tissue concentrations tend to follow seasonal cycles, which may be related to variations in lipid content, spawning cycles, or environmental flux (Jovanovich and Marion 1987; Maruya et al. 1997; Miles and Roster 1999).

Bioaccumulation values can be useful for gauging the degree to which contaminants have been bioaccumulated across species and locations. Many studies (e.g., Lake et al. 1990; Bartell et al. 1998) have shown that biota-to-sediment accumulation factor (BSAF) values at steady state reduce the vari-

ability inherent with simple bioaccumulation factors (BFs) based on the ratio tissue concentration to sediment concentration. In the BSAF approach, the ratio is derived using a sediment concentration normalized to organic carbon and a tissue concentration normalized to its lipid content. However, there is a substantial amount of data, particularly in the freshwater literature, demonstrating that the BSAF approach is not adequate to describe the extent a compound will accumulate (e.g., Van Hoof et al. 2001). The recent appendix to the USEPA (2000) bioaccumulation guidance document shows BSAF values exceeding 50 for some chlorinated pesticides. Such large numbers suggest that factors other than the amount of organic carbon in the sediment are responsible for controlling the bioaccumulation, and simple predictions of BSAF should be used with caution. Additionally, there has been a tendency to misuse the BSAF. The BSAF approach was never intended for certain applications such as the normalization of fish tissue concentrations or the assessment of bioaccumulation of metals. For fish, this approach may be applicable, depending on the compound, species, and its site fidelity, and on the degree to which different matrices are characterized. In general, this application ignores the impact of trophic transfer for some compounds even when the primary source is sediment. Finally, it has been shown that normalizing to the amount of organic carbon, which presumably governs bioavailability, can be ineffective in improving the predictive ability of the empirically based SQGs (e.g., Ingersoll et al. 1996; Chapters 4 and 12).

In addition to the difficulties in estimating tissue residue concentrations from sediment chemistry data, interpreting the significance of these predicted residue concentrations has also been problematic. Interpretation of tissue residue concentrations has historically been limited to comparison to a fixed standard (e.g., US Food and Drug Administration [USFDA] action level or comparison to reference). Recent efforts on the part of the US Environmental Protection Agency [USEPA] and US Army Corps of Engineers [USACE] have helped to establish more formalized guidance for interpreting tissue residue data. Using such tools as the critical body residue (CBR) model for nonionic organics (McCarty and Mackay 1993), the Environmental Residue Effects Database (ERED) (USACE/USEPA 2002a) and the Jarvinen and Ankley (1998) database have enabled a more thorough assessment of the potential effects associated with measured tissue concentrations. However, because of the paucity of residue-effects data and the need to assess potential effects to higher trophic levels, extrapolations between compounds and species and applications of models are sometimes employed to assess the potential effects of bioaccumulation. Tissue residue data have been used for establishing trophic transfer

models to estimate the potential for contaminant exposure from sediments on higher-level organisms (e.g., Thomann et al. 1992; Thomann and Komlos 1999). It may be possible to link these tools for purposes of estimating sediment concentrations expected to be deleterious to organisms through bioaccumulation. For example, when tissue concentrations associated with adverse effects have been identified, it may be possible to use site-specific BSAF values to calculate sediment concentrations that would be expected to produce a toxic tissue concentration (Meador et al. 2002a). Currently, there appear to be 2 potential types of SQGs that can be developed for assessing potential effects of contaminant bioaccumulation from sediments, namely direct guidelines based on tissue residue effects data and guidelines that incorporate the indirect effects through the action of trophic transfer. The primary concern in developing meaningful guideline values is addressing the uncertainty associated with either approach. Any bioaccumulation-based SQG value must possess a clear ability to predict the bioaccumulation potential for contaminants from sediments as well as the subsequent effects of that exposure in the receptors of concern. The following sections focus on questions fundamental to understanding and developing SQG values predictive of bioaccumulative effects.

Are Results from Laboratory Bioaccumulation Studies Predictive of Tissue Residues in Field-Collected Organisms?

One important question concerns the fidelity between laboratory and field assessment of bioaccumulation. Many bioaccumulation tests are performed in the laborarory with the assumption that the results will reflect the bioaccumulation potential of organisms in the field. It is clear that the conditions under which toxicity tests are normally performed in the laboratory reflect only a snapshot of the possible exposure conditions for organisms in the field. However, if the laboratory conditions are varied sufficiently, it may be possible to predict the range of results expected in the field (e.g., Landrum et al. 2001). Field-collected BSAF values have generally followed predicted values (Wong et al. 2001). It is also important to note that bioaccumulation studies are generally focused on benthic infauna (i.e., animals with limited mobility and in direct contact with sediment). Consequently, the application of BSAFs to fish is probahly not meaningful because of variahle degrees of site fidelity and the potential impact of trophic transfer.

In Table 11-1, several studies that examined hioaccumulation in invertebrates in the laboratory and field are listed. In general, a comparison of values for a

given compound or class of compounds shows substantial variability; however, when means are considered, relatively consistent patterns emerge. For the PAHs, BSAFs are consistently in the 0.01 to 0.5 range across species and are not appreciably different between those determined in the laboratory and those measured in field-collected organisms. The BSAFs for chlorinated hydrocarbons (CHs) are generally much higher than those for the PAHs, ranging from 0.2 to 5. For both groups, values much lower, and sometimes much higher than these, have been reported. The lower values generally include the more highly hydrophobic compounds or those that can be metabolized. If total polychlorinated biphenyls (PCBs) are considered, it appears that there is very little difference between the laboratory and field-based BSAF values (Table 11-1), leading to the conclusion that lab-generated BSAFs can be predictive of those in the field.

The differences between PAHs and CHs are highlighted in studies that examined these compounds in the same species and sediments (Landrum and Faust 1991; Hickey et al. 1995; Meador et al. 1995; Meador, Adams et al. 1997). The differences between PAHs and CHs may be due to the metabolic transformation of PAHs by many of the species tested and the lack of metabolism for PCBs by most species. Other factors for the observed differences include, but are not limited to, differences in the bioavailable fraction for each compound, differential toxicokinetics for chemicals, and insufficient time for accumulation.

In general, many studies have demonstrated that BSAFs for PAHs in benthic invertebrates appear to occur at values approximating 1 order of magnitude below those BSAF values (generally ranging from 1 to 4) expected at equilibrium (Bierman 1990; Di Toro et al. 1991; Boese et al. 1995). Without detailed analysis, it is generally not possible to determine which factors (e.g., reduced exposure time, metabolism, reduced bioavailability) are most important for this observation. When nonmetabolized, nonionic, organic compounds are examined, measured BSAF values are often close to expected values (Bierman 1990; Tracey and Hansen 1996; Wong et al. 2001), but deviations can occur (USEPA 2000).

One analysis of BSAFs from the literature found no differences between laboratory-and field-exposed benthic invertebrates for PAHs (USACE/USEPA 2002). The mean and standard deviation (sd) PAH BSAF values for laboratory-exposed and field-exposed benthic invertebrates were 0.34 (0.95), n = 167, and 0.36 (0.98), n = 183. The median values for each group were identical (= 0.077). Based on Dunn's test on medians, the median BSAF values for CHs were significantly different (USACE/USEPA 2002). The lab-exposed benthic

Table 11-1 Bioaccumulation factors for polycyclic aromatic and chlorinated hydrocarbons (PAHs and CHs) by invertebrates

Species	Feeding type[a]	Area, type[b]	Compounds[c]	Total conc (ng/g) dry	BAF (dry wt)	BSAF (range)	BSAF (mean)	Time (d)	Ref
Abarenicola pacifica	DF	Northwest USA, LS	3 PAHs			1.5–3.7	2.4	60	Augenfield et al. 1982
Arenicola marina	DF	Netherlands, LF	7 PAHs	370–3;100	0.76			60–90	Kaag et al. 1997
Armandia brevis	DF	New York USA, LF	Sev CHs[d]			0.2–6.3	0.44[e]	10	Meador et al. 1997a
Armandia brevis	DF	New York USA, LF	24 PAHs			0.002–0.9	0.18	10	Meador et al. 1995
Austrovenus stutchburyi	FF	No. New Zealand, F	Sev CHs[d]	1.2–16	1.2–51.5	0.2–3.8	1.45[e]		Hickey et al. 1995
Austrovenus stutchburyi	FF	Northern New Zealand, F	9 PAHs	9–47	0.01–0.58	0.002–0.05	0.04		Hickey et al. 1995
Chlamys septemradiata	FF	Norway, F	12 PAHs	75–304		0.007–0.43	0.11		Naes et al. 1999
Corophium volutator	Detr	Netherlands, LF	8 PAHs			0.5–1.7		25	Kraaij et al. 2001
Coullana sp.	Omn	Louisiana USA, LS	fluoranth			0.22–0.67	0.43	1	Lotufo 1998
Diporeia spp.	DF	Lake Michigan USA, LS	DDT			0.03–0.31	0.15	28	Lotufo et al. 2001
Eohaustorius washingtonianus	Detr	Northwest USA, LF	16 PAHs		0.1–0.45			7	Varanasi et al. 1985
Hyalella azteca	Omn	Lake Michigan USA, LS	DDT			0.44–2.1	0.97	28	Lotufo et al. 2001
Leptocheirus plumulosus	Detr, FF	Chesapeake Bay USA, LS	fluoranth				0.32	26	Kane Driscoll et al. 1998
Lumbriculus variegatus	DF	Green Bay, Wisconsin USA, LF	Total PCBs			0.21 - 1.35	0.84[e]	30	Ankley et al. 1992
Macoma balthica	DF, FF	Chesapeake Bay USA, LS	chlor. pesth			0.17–0.78	0.17	12	[illegible]

Table 11-1 contd

Species	Feeding type[a]	Area, type[b]	Compounds[c]	Total conc (ng/g) dry	BAF (dry wt)	BSAF (range)	BSAF (mean)	Time (d)	Ref
Macoma inquinata	DF, FF	Northwest USA, LS	3 PAHs			0.6–2.4	1.3	60	Augenfield et al. 1982
Macoma nasuta	DF	Southern California USA, LF	11 PCBs			0.21–2.1[f]	0.91	28	Ferraro et al. 1991
Macoma nasuta	DF, FF	Oregon USA, LS	PCBs, HCB		0.9–30.3	0.06–2.0	1.2[e]	119	Boese et al. 1995
Macoma nasuta	DF, FF	Northwest USA, LF	16 PAHs		0.06–0.19			28	Varanasi et al. 1985
Macoma nasuta	DF, FF	Los Angeles USA, LF	5 PAHs			0.2–1.0	0.4	28	Ferraro et al. 1990
Macomona liliana	DF	Northern New Zealand, F	Sev CHs[d]	1.3–47	3.1–61.3	1.4–23.2	3.5[e]		Hickey et al. 1995
Macomona liliana	DF	Northern New Zealand, F	9 PAHs	18–203	0.09–1.7	0.04–0.13	0.10		Hickey et al. 1995
Mya arenaria	FF	Chesapeake Bay USA, LS	chyr, naph			nd		12	Foster et al. 1987
Nephtys incisa	Omn	Northeast USA, F	Total PCBs			3.2–4.3	3.8		Lake et al. 1990
Nereis virens	Omn	California and New Jersey USA, LF	fluoranth			0.8–3.3		15	Brannon et al. 1993
Oligochaetes	DF	Green Bay, Wisconsin USA, F	Total PCBs	≈ 1800		0.87–2.59	0.87[e]		Ankley et al. 1992
Palaemonetes pugio	Omn	Louisiana USA, LS	BaP, phen			nd–1.6	0.23	14	Mitra et al. 2000
Polychaetes (several spp)	DF, Omn	San Francisco USA, F	18 PAHs	310–1790		0.04–2.0	0.2		Mayrua et al. 1997
Potamocorbula amurensis	FF	San Francisco USA, F	18 PAHs	130–860		0.6–5.4	0.3		Mayrua et al. 1997
Rangia cuneata	FF	Louisiana USA, LS	BaP, phen			nd–1.5	0.42	14	Mitra et al. 2000

Table 11-1 *contd*

Species	Feeding type[a]	Area, type[b]	Compounds[c]	Total conc (ng/g) dry	BAF (dry wt)	BSAF (range)	BSAF (mean)	Time (d)	Ref
Rhepoxynius abronius	Omn	New York USA, LF	Sev CHs[d]			0.02–0.95	1.6[e]	10	Meador et al. 1997a
Rhepoxynius abronius	Omn	New York USA, LF	24 PAHs			0.001–0.5	0.052	10	Meador et al. 1995
Rhepoxynius abronius	Omn	Northwest USA, LF	16 PAHs		0.09–0.5			7	Varanasi et al. 1985
Schizopera knabeni	DF	Louisiana USA, LS	fluoranth			0.51–0.80	0.62	1	Lotufo 1998
Stichopus tremulus	DF	Norway, F	12 PAHs	237–797		0.004–0.67	0.18		Naes et al. 1999
Streblospio benedicti	DF	South Carolina USA, F	3 PAHs	860–2000	0.2–1.4	0.08–0.4	0.22		Ferguson and Chandler 1998
Tapes japonica	FF	San Francisco USA, F	18 PAHS	95–450		0.007–2.7	0.15		Mayrua et al. 1997
Yoldia limatula	DF	Northeast USA, F	Total PCBs			4.1–4.8	4.4		Lake et al. 1990

[a] DF = deposit feeder; FF = filter feeder; Omn = omnivore; Detr = detrivore

[b] Where found or where research was conducted, type of exposure (F = samples from field; L = exposures in lab to field-contaminated sediment; LS = exposures in lab to spiked sediments)

[c] Fluoranth = fluoranthene; phen = phenanthrene; BaP = benzo[*a*]pyrene; BA = benz[*a*]anthracene; HCB = hexachlorobenzene; CH = chlorinated hydrocarbons). All analyses conducted with whole organisms or soft tissue of clams. Mean BSAFs includes all compounds and locations studied.

[d] PCBs, DDTs, and chlorinated pesticides (e.g., lindane, dieldrin, chlordane). A conversion factor of 5 was used for wet weight to dry weight concentrations.

[e] Total PCBs

[f] 0 to 2 cm depth

invertebrates exhibited a higher median BSAF of 0.85, $n = 280$ compared to field-exposed benthic invertebrates of 0.32, $n = 414$. The mean (sd) values were closer at 1.4 (1.7) for lab-exposed invertebrates and 1.2 (2.4) for field-exposed invertebrates. The field-exposed invertebrates were expected to have the higher BSAFs resulting from increased bioaccumulation time. However, these results may indicate that field conditions are more heterogeneous than those found in the laboratory and may be influenced more by external factors such as fresh food or alternate sources of contaminants. The PAH results may also indicate the same mechanism. However, interspecies differences in metabolism may obscure any such pattern.

A few studies have addressed the laboratory versus field question directly. Ankley et al. (1992) compared BSAF values for oligochaete worms exposed to PCBs in sediment. They measured concentrations in worms from the field and then exposed *Lumbriculus variegatus* in the laboratory to these same sediments for 30 days. For 3 of the PCB congener classes (dichloro, heptachoro, and ocrachloro), oligochaeres from the field exhibited significantly higher BSAF values than those exposed in the laboratory. Additionally, Brunson et al. (1998) found good relationships between laboratory-exposed oligochaetes and field-collected organisms for PAHs. Ninety percent of the paired PAH concentrations between laboratory and field samples fell within a factor of 3. Oliver (1987) reached a similar conclusion with regard to persistent organic compounds while examining BAFs in laboratory exposures and field collections of oligochaetes. Ingersoll et al. (2003) reported that concentrations of DDT, DDD, and DDE, and PAHs measured in native oligochaetes which were collected at the same time that sediment was collected from the field were similar to the steady-state concentrations estimated from the laboratory exposures with the oligochaete *L. variegatus*. However, concentrations of low-K_{ow} PAHs in native oligochaetes were biased higher than the steady-state concentrations estimated from the laboratory exposures with *L. variegatus,* while concentrations of high-K_{ow} PAHs in native oligochaetes were biased lower. In another laboratory study, Landrum et al. (2001) examined the BSAF of PCB congeners in *Diporeia* spp. and found that the BSAF was not constant with log K_{ow} as suggested from EqP theory. They concluded, however, that field BSAF values could be predicted from laboratory data, provided the organism size and exposure temperature were taken into consideration.

Studies in which organisms are placed in cages or on racks and left in the field for a predetermined period of time can be a useful way to assess bioaccumulation. These techniques are useful because they allow researchers to get information from species not found at the site, compare results across sites for

the same species, and control several temporal and spatial factors that potentially affect bioaccumulation. The technique of placing bivalves at a site has been used for years to assess bioaccumulation. Mussels are commonly used f this type of experiment because of their high rate of water filtration and low biotransformation capacity. Bivalves can also be sampled from the field and assessed for bioaccumulation (e.g., Mussel Watch program); however, in situ placement allows greater control of important variables. One drawback to us ing mussels to monitor bioaccumulation is that they do not interact directly with the sediment and may exhibit much less bioaccumulation than other benthic species.

Other species, including benthic invertebrates and fish, have been placed in cages left on site for various periods of time to monitor contaminant accumulation under more realistic field exposure conditions. These in situ toxicity tests also can be used to measure biological effects such as mortality or reduced growth. Some of the biggest challenges facing these studies include the selection of comparable reference sites, effects due to caging, loss of experimental units, and variability in environmental parameters that can affect toxicokinetics and organism health (e.g., temperature, food, dissolved oxygen pH). Details for caging and transplant studies can be found in the literature (e.g., Chappie and Burton 1997; Salazar and Salazar 1998; Forrester et al. 2003).

What Factors Affect Assessments of Bioaccumulation?

Several factors can affect the results in bioaccumulation assessments, regardles of the origin of the exposure source (field contaminated or spiked) or location (field versus laboratory). A number of these factors and their potential effects on bioaccumulation are discussed below.

Route of exposure: Field versus laboratory sources

Bioaccumulation of contaminants in the field by aquatic organisms results from exposure to multiple potential sources. For benthic organisms, these include sediment, sediment pore water, ingested particles including fresh detritus and bacteria, and overlying water. Many of these sources are out of chemical equilibrium with each other. Tissue residue concentrations provide a measure of contaminant exposure from all potential sources and metabolism by the organism. Further, the concentration in the environment of the

organism may be different from that taken for analytical measurements, which would impact attempts to establish relationships between single sources and measured bioaccumulation. When exposure occurs in the laboratory setting, the resultant accumulation may not reflect the environmental measurements because the sources are not reflective of those in the environment. The experimental conditions, such as temperature and population structure, that affect the physiology and thus the toxicokinetics are not reflected in the laboratory environment. The exposures in the laboratory tend to be snapshots of specific conditions that may be found in the field but cannot reflect the overall exposure of organisms over temporal and spatial scales with multiple sources of exposure.

Accumulation of nonionic hydrophobic compounds is generally passive and is driven by the chemical activity of the source and sink compartments. However, the rate may well be dictated by the route and volume of source compartment that the organism experiences. Uptake from water is generally accomplished by ventilation over the gill structure; however, diffusion through the integument may also contribute to tissue concentrations (Landrum and Stubblefield 1991). Ingestion of prey organisms, detritus, and sediment is also important for accumulation. In the short term (before steady state), the degree to which each route contributes to the total body residue is difficult to determine without well-designed experiments. According to EqP theory, when all phases are in equilibrium, the route of uptake is immaterial because no matter which route dominates, the resulting tissue concentration is always the same (Chapter 13). However, systems are rarely in chemical equilibrium such that EqP would apply (Lee 1990). For instance, the overlying water does not attain expected equilibrium with surficial sediments for PCB partitioning in Lake Michigan (P.F. Landrum, personal communication NOAA GLERL, Ann Arbor, MI), and while individuals of the amphipod *Diporeia* spp. are at steady state with the sediment concentrations based on the toxicokinetics and field measurements for PCBs, the BSAF values deviate substantially from expected EqP theory, ranging from approximately 0.2 to greater than 10 (Landrum et al. 2001).

Bioaccumulation of metals is more complicated than that of organic compounds. The control of water concentrations is complex and often not correlated in a linear fashion to the ligands that control their solubility. Many studies indicate that porewater concentrations are controlled by AVSs, which in turn are correlated to the degree of toxicity (Ankley et al. 1996) and presumably the amount bioaccumulated. Other studies, however, have demonstrated

that dietary uptake may also be an important route for bioaccumulation of metals (Weeks and Rainbow 1993; Roy and Hare 1999).

The exposure matrix can be an important determinant for bioaccumulation, especially under nonequilibrium conditions. Benthic species are likely to exhibit higher tissue residues because they often ingest sediment and are exposed to pore water. There are a variety of feeding modes for benthic species, including but not limited to sediment ingestion (selective and nonselective), detritus feeding, predation, and filter feeding. Each of these modes may have an impact on the degree that the organism is exposed to contaminants and final bioaccumulation values, especially if disequilibrium prevails among water, tissue, and sediment. Some infauna are exposed to pore water, and some build tubes and pump overlying water through their burrows. Studies that compare different species under identical conditions can be very informative when the bioaccumulation potential is being determined.

A number of studies have examined the mode of feeding by invertebrates in relation to bioaccumulation (Foster et al. 1987; Hickey et al. 1995; Meador et al. 1995; Kaag et al. 1997). For example, Foster et al. (1987) demonstrated large differences in tissue concentrations between 2 clams, one a deposit feeder and the other a filter feeder (Table 11-1). Another study comparing *Rhepoxynius abronius* (an infaunal amphipod that does not ingest sediment) and *Armandia brevis* (an infaunal nonselective deposit-feeding polychaete) found similar accumulations of LPAHs by the 2 species but substantially more accumulation of HPAHs by the polychaete (Meador et al. 1995). Because these were lab toxicity tests, the amphipod likely had little prey available, which led to the conclusion that deposit and nondeposit-feeding infaunal invertebrates will acquire most of their body burden of LPAHs through pore water, regardless of feeding strategy; however, ingestion (of sediment or food) may be the dominant route of uptake for hydrophobic compounds exceeding a log K_{ow} of approximately 5.5. If sufficient prey were available to *R. abronius* in this experiment, it is likely that BSAF values for the HPAHs would have been higher.

Temporal issues

The amount of time allowed for bioaccumulation can have a large effect on the tissue residues and the degree to which the organism reaches steady state. Several studies have examined the temporal aspects of bioaccumulation. Oliver (1987) found in a study of 37 CHs that oligochaete worms reached steady-state tissue residues within 2 weeks for most compounds. Accumulation of PAHs and DDT from sediment by the oligochaete *L. variegatus* typically

reached steady state in 14 to 28 d during 56-d laboratory exposures (Ingersoll et al. 2003). Several of the more hydrophobic compounds ($\log_{10} K_{ow} > 6.0$) exhibited relatively long half-lives, indicating that steady state would take longer. Most bioaccumulation studies are conducted for 28 d because of the recommendations and research by Lee et al. (1993) showing this period of time to be sufficient for many infaunal benthic species and chemicals to reach at least 80% of steady-state tissue concentrations. At least 1 study has found that very small benthic invertebrates achieve steady state within hours to a few days when exposed to sediment-associated fluoranthene (Lotufo 1998), which may be related to their large surface area to volume ratio.

The time to achieve steady state is dictated by the magnitude of the elimination rate constant. The elimination rate constant can be substantially influenced by the lipid content of the organisms. For instance, the elimination rate of *Hyalella azteca* for fluoranthene is relatively rapid, such that steady state would be achieved in approximately 30 h, while the more lipid-rich *Diporeia* has substantially slower rates of elimination that would not result in achievement of steady state until upward of 35 to 130 d (Kane-Driscoll et al. 1997). While some of the difference was attributed to the biotransformation capability of *H. azteca,* the majority of the effect was thought to result from the very high lipid content of *Diporeia.*

Seasonality

Organisms show changes in their lipid content with the season, and these changes can result in either increased or reduced storage capacity, depending on whether the organisms are accumulating or consuming lipid for energy or for transfer to offspring. The influence of lipids on bioaccumulation and trophic transfer of organic contaminants has been reviewed by Landrum and Fisher (1998). Another study was designed to specifically examine the seasonality of PCB bioaccumulation by *Diporeia* spp. (Robinson et al. 2000). While seasonality was not strong, that is, BSAF values varied only within a factor of 2 for a specific congener, 2 factors appeared to dominate the limited seasonal trends in the data. One important factor was the accumulation of lipids with ingestion of the spring diatom bloom resulting in apparent concentration dilution in the tissues, which reduced the BSAF. A second factor was alterations in the organic carbon–normalized PCB concentration in source. Alterations also occurred in the organic carbon–normalized PCB concentrations in the source. This dilution of the source with fresh carbon had the net effect of reducing the overall source concentration, thus elevating the BSAF. Similarly, the influence of lipids and the spawning cycle on the bioaccumulation of PCB

in *Mytilus edulis* was found for exposure in Buzzard's Bay, resulting in a 2- to 4-fold difference in the bioaccumulation (Capuzzo et al. 1989). Other factors such as organism feeding behavior, food composition, and the population size distribution of food sources can also influence seasonal exposure to contaminants, both through changes in the rate of feeding and in the choice of food.

Uptake efficiency

Some studies indicate that uptake efficiency generally declines with increasing chemical hydrophobicity, which may be due to a combination of slow desorption kinetics and short residency time in the gut. Additionally, because the role of metabolism often is not addressed, the apparent uptake efficiency may be underestimated because of the effective loss of the parent compound. For some compounds, it has been demonstrated that the rates and efficiencies of uptake vary slightly over K_{ow} and are therefore not strongly linked to chemical hydrophobicity (McKim et al. 1985; Bender et al. 1988; Landrum et al. 2001). Other studies demonstrate that aquatic organisms exhibit strong relationships between the rate of uptake and log K_{ow} (e.g., Landrum 1988).

In order to avoid confounding the estimate of uptake efficiency for compounds that are metabolized, the parent compound plus metabolites should be determined. For example, research that examines uptake and elimination kinetics would be needed to better assess uptake efficiency of PAHs for the different routes of uptake, especially the dietary route. These data would help greatly in predicting bioaccumulation from different environmental matrices.

Metabolism

Metabolism of xenobiotic compounds is a crucial factor in determining and predicting bioaccumulation. Some hydrophobic organic compounds are poorly metabolized by invertebrates and fish (e.g., PCBs), while others are biotransformed. These processes, however, vary widely among different taxa. Elements are not metabolized but are often rendered less toxic by complexation with metallothionein or are incorporated into granules, shell, or bone. Among benthic invertebrates, metabolism of PAHs can be highly variable, even within taxonomic groups. For example, large differences in metabolic transformation of PAHs were found for different species of polychaetes by Kane-Driscoll and McElroy (1996). Among crustaceans, some species such as *R. abronius* have relatively active P450 systems (Reichert et al. 1985), and others such as *Chironomus tentans* show high biotransformation while the P450 system has not been directly evaluated (Lydy et al. 2000). Conversely, other crustaceans, such as the American lobster (*Homarus americanus*) (Foureman

et al. 1978; Bend et al. 1981) and *Diporeia* spp. (Landrum 1988), have very limited biotransformation capabilities for some compounds such as PAHs. In general, mollusks accumulate high levels of contaminants, including PAHs because of their high rate of filtration and low metabolic capacity at steady state despite having low lipid contents (Livingstone 1994).

Sometimes the assessment of metabolism in determining bioaccumulation is not straightforward. For example, large differences were observed in BAF values for 2 infaunal amphipods (*Eohaustorius washingtonianus* and *R. abronius*) and a deposit-feeding clam (*Macoma nasuta*) exposed to benzo[*a*]pyrene (Varanasi et al. 1985). The amphipods exhibited higher BAFs than the clam, although their metabolic capacity for this compound was much greater. Several factors may explain these unexpected results, including a higher rate of uptake leading to higher body burdens or exposure to additional sources of contaminant such as ingestion. Additionally, the clam may have simply interacted more with overlying water or it may have closed its valves, thus reducing contaminant uptake. The BAFs for the amphipods at 7 d of exposure were several-fold higher than the BAFs observed for the clam at 28 d, a time sufficient for near steady-state accumulation to occur (Lee et al. 1993).

Aging of Sediment-Associated Contaminants

A limited number of studies have identified the effects of aging and the change in bioavailability of a contaminant with increasing contact time between the contaminant and sediment particle (e.g., Landrum 1989). The impact seems to be larger and more rapid with compounds that are less hydrophobic, likely because they fit better into sediment particle pores. The difficulty has been to assess the impact of this mechanism on field samples that presumably have long contact times. In the laboratory, long contact times have been used as a way to ensure equilibrium prior to any bioaccumulation testing. This allows for better comparisons with the field condition (USEPA 2000). Studies associating the rate and extent of desorption from the rapidly desorbing fraction to the extent of biodegradation and bioaccumulation of contaminants have shown that the rapidly desorbing fraction, whether in laboratory-dosed sediment or in native sediments, correlates with bioavailability (Cornelissen et al. 1998; Kraaij et al. 2001). This approach is new and the data are sparse; however, this does suggest an approach for moving from total contaminant concentration to a practical measure of the bioavailable contaminant without necessarily performing toxicity tests. Recent work with organic contaminants suggests that it is not the fraction of rapidly desorbing

contaminant that is responsible for bioavailability; rather it is the flux (i.e., the concentration in the rapidly desorbed fraction times the desorption rate constant) off the particles that dictates the bioaccumulation (Kukkonen et al. 2001). Research focused on the role of desorption kinetics shows great promise in providing a method for evaluating the bioavailability through chemical means, but more research is required.

Physical and chemical factors

The amount of organic carbon for sorbing organic contaminants and the amount of ligands for binding metals are widely recognized as modifying the bioavailability of sediment-associated contaminants. The quality of organic matter is also recognized as critical to the bioavailability of organic contaminants. The polarity of organic matter has been demonstrated to alter the rate of accumulation of PAHs (Landrum et al. 1997). However, the same work showed that differences in bioaccumulation for PCBs were most readily attributed to total organic carbon content and polarity was not a major factor. Thus, it appears that the chemical characteristics of the contaminant and the manner in which it interacts with the sediment organic matter are critical for dictating bioavailability. Because of selective feeding and the potential role of feeding on the bioaccumulation of contaminants, the uneven distribution of the contaminants on particles of varying sizes could have a large influence on the bioaccumulated fraction. The bioaccumulation and assimilation efficiency for PAH versus PCB congeners were tied to this unequal distribution and the selective feeding of *Diporeia* spp. (Harkey, Lydy, et al. 1994). In the field, there is some indication that such differential distribution of compounds among particles of differing sizes does exist (Umlauf and Bierl 1987; Evans et al. 1990; Pierard et al. 1996; Van Hoof and Eadie 1999).

For metals, the extent of binding to ligands, particularly AVS, has been demonstrated to reduce bioavailability (Di Toro et al. 1990; Chapman et al. 1998; Chapter 13). However, many other ligands can participate in the reduction in bioavailability, depending on the composition of the system (Newman and Jagoe 1994). From a practical perspective, it appears that the porewater concentration more accurately predicts toxicity (e.g., Ankley et al. 1993). It is clear, however, that environmental exposure to metals remains complex, and additional work to establish a useful practical approach to evaluating metal bioavailability remains to be developed.

Contaminant source

The bioavailability of some contaminants can vary considerably within a limited region, likely resulting from differences in sources. For example, the source for PAH exposure varies widely, with some sites containing mainly petroleum-based PAHs while others are contaminated with combustion PAHs. Several studies have shown reductions in water or tissue concentrations of PAHs as they may relate to PAH type and source (Farrington et al. 1983; McGroddy et al. 1995; Meador et al. 1995; Maruya et al. 1997; Naes et al. 1999). For example, PAHs from different combustion sources (e.g., soot, coal, or an aluminum smelter) may produce very different bioaccumulation patterns. Likewise, fresh petroleum would likely result in different bioaccumulation patterns compared to weathered petroleum because of proportional change among congeners. One interesting study (Naes et al. 1999) examined BSAF values in 3 different benthic invertebrates and found a gradient of decreasing values towards an aluminum smelter. Depending on the species, BSAFs decreased from 5- to 10-fold from one end of a fjord to the other end where the smelter was located and may be related to the types and sources of PAHs found along the gradient. Another srudy proposed that the lower PAH BSAF values observed by Landrum (1988) for the amphipod *Diporeia* spp., a species that is not able to biotransform PAHs, was due to the importance of soot on the partitioning of PAHs in sediment systems (Van Hoof et al. 2001). The impact of soot carbon has also been implicated in limiting the bioaccumulation of PCBs in Ashtabula Harbor, Ohio, USA (Pickard et al. 1998).

Organism behavior

Organism activity can also affect the sorption dynamics of contaminants associated with sediment. For example, the suite of organisms nearby the target species in the laboratory or the field may have a significant effect (approximately a factor of 2) on the amount of a contaminant that is bioaccumulated (Schuler et al. 2002). Bioturbation by organisms can increase contaminant concentrations in overlying water, which may be an important factor in assessing bioaccumulation for some species. For example, McElroy et al. (1990) demonstrated that the presence of a tubiculous polychaete (*Nereis virens*) can enhance the flux of sediment-sorbed benz[*a*]anthracene to the water column. This increased flux to the water column could elevate tissue concentrations in those animals that take in contaminants through gill membranes by ventilating overlying water. This was also confirmed by Ciarelli et al. (1999), who found that bioturbation by 1 species enhanced bioaccumulation in another. In this study, the authors showed a linear relationship between fluoranthene

in *Mytilus edulis* and amphipod *Corophium volutator* density. Additionally, benthic organisms can inhibit the desorption of chemicals into water. For example, it has been observed that oligochaete fecal pellets can dramatically reduce the amount of compound that will desorb into water (Karickhoff and Morris 1985). These mechanisms are important because severe alterations to sediment–water partitioning and equilibrium can occur, thus confounding comparisons of bulk sediment and tissue concentrations for bioaccumulation assessment.

Some species may change their behavior as tissue residues approach toxic levels. In some cases, animals may exhibit adverse effects and alter their rates of ingestion or ventilation, causing tissue concentrations to increase or decrease dramatically. This phenomenon has been observed by Landrum et al. (1994) and Meador and Rice (2001). Exposure avoidance is another approach for reduction in observed toxicity (Kukkonen and Landrum 1994). Basing the toxicity on the concentration in the organism, therefore, rather than on the exposure environment, allows improved interpretation of the toxicity data.

Species differences

In general, it is believed that species within a given taxonomic family will exhibit similar responses to toxicants (Suter and Rosen 1986). Several studies have shown that relatively closely related species can exhibit very different toxicity responses as a result of differential bioaccumulation. For example, Meador, Krone, et al. (1997) found that 2 species of amphipods from closely related families (Superfamily Haustorioidea) exhibited LC50s to tributyltin (TBT) that differed by 14-fold in 10-d water exposures. When compared to *E. estuarius, R. abronius* was the more tolerant species. Even larger differences were observed in long-term sediment exposures with *R. abronius* and *E. washingtonianus*, a species that responds identically to TBT as *E. estuarius* (Meador, Krone, et al. 1997). When these same species were exposed to Cd in water, *R. abronius* was the more sensitive of the two, exhibiting an LC50 that was 10× lower (ASTM 2004). Interestingly, another study found that these 2 species responded similarly to sediments contaminated with PAHs (Pastorok and Becker 1990).

Certainly, the use of body residue as a dose metric suggests that for nonpolar narcosis (anesthesia), the toxic response for acute mortality has a narrow range for fish (McCarty and Mackay 1993). The difference for this mechanism of action between species depends strongly on the lipid content of the organisms (van Wezel and Opperhuizen 1995). However, the relative species response may well depend on the mechanism of toxic action. For specific acting chemi-

cals, larger variability can be expected because of the need for toxicant to fit to a specific receptor that is likely different for different species, even closely related ones. In a review by Barron et al. (2002), the issue of species differences and particularly differences in mechanisms of action contributes significantly to the observed body residue to produce a toxic response. Thus, toxicity tests or field assessments with only a few species may underestimate the toxicity potential of a given sediment.

Lipid content

Lipid content in organisms is an important factor for assessing bioaccumulation of nonionic hydrophobic compounds. Even though the lipid content in many invertebrates approximates 5% (dry weight) (Boese and Lee 1992), many exceptions occur (e.g., *Diporeia*; Landrum and Nalepa 1998) and can range up to 50%. The ability of the lipid to sequester contaminants away from the site of toxic action is critical to the observed response and has led to the hypothesis of the survival of the fattest (Lasiter and Hallam 1990). To adequately test the hypothesis that lipids control the bioaccumulation of nonionic hydrophobic compounds, lipid content would need to be varied for a given species and toxicokinetic rates measured in order to ensure that they are comparable between groups. One study (Bruner et al. 1994) found that high-lipid zebra mussels had greater BCFs and faster uptake kinetics for highly hydrophobic compounds.

For some hydrophobic compounds, lipid may not be important for bioaccumulation. For example, the bioaccumulation of TBT was found to be far more extensive than that predicted by its K_{ow}, leading to the conclusion that lipid does not play a role in determining tissue residues for TBT (Meador 2000). It is expected, however, that for a given whole-body tissue concentration, the magnitude of the toxic response from TBT may be a function of the organismal lipid content (Meador 1993, 2000), although the data are not sufficient for a rigorous test of this hypothesis.

Spatial issues

The relationship between the measured contaminant concentrations found in sediment and those found in the organisms may be complicated by sampling errors. If the matrix that produces the exposure is not the matrix analyzed, then there will be a disconnect in the relationship between concentrations in sediment and the organism. For instance, Lee (1991) points out that it is typical for analytical chemists to select the top 2 cm of sediment, while organisms may feed on the very surficial material (fresh-falling detritus, e.g., *Macoma*) or

may feed at depth (e.g., oligochaete or polychaete worms). Additional issues include spatial variability and could include temporal variability for labile co taminants (Holland et al. 1993; Sarda and Burton 1995). The extent of the variability can result in variation in exposure conditions and in a poor match between the measured exposure concentration and that accumulated by the organism, resulting in additional variability in observed relationships.

Organism size

Clearly, even within a species, there are characteristics that relate organism si: to the exrent of accumulation. In some organisms, the mode of feeding chan es as the organism metamorphoses from larval to juvenile to adult forms. Further, feeding rates, filtering rates, etc., are allometric, and they change the resultant exposure and toxicokinetics of organisms. For instance, in *Diporeia* spp., the accumulation of PCB congeners is greater in small organisms compared to larger ones, primarily because of changes in the respiration relationship to contaminant accumulation (Landrum and Stubblefield 1991) and th higher feeding rate for smaller organisms (Lozano et al. 2003). The resultant bioavailability in small organisms is not due to preferential ingestion of small particles because distribution to smaller particles with higher organic carbon apparently reduces bioavailability (Harkey, Lydy, et al. 1994). The result is that small *Diporeia* show greater accumulation in the field compared to even older, larger organisms (P.L. Van Hoof, personal communication, Monrovia, MD). These higher rates are manifest in larger BSAF values for smaller amph pods.

Habitat: Laboratory versus field

Biomass loading of the experimental systems in the laboratory may not reflec that in the field. This may create variation in the observed bioaccumulation of contaminants. For the oligochaete *L. variegatus*, increasing the biomass loading relative to the amount of organic carbon increased the exposure of th organisms (Kukkonen and Landrum 1994). Additionally, differences in orgar ism abundance can have an effect on sediment geochemistry and the amount of contaminant available for uptake.

Feeding

The feeding behavior of the species can be important in assessing its bioaccumulation of sediment-associated contaminants. Benthic species exhibit several feeding strategies, some of wbich maximize their exposure to contaminants and others that allow very little exposure. When an organism is in contact

with sediment but does not ingest sediment, equilihrium or steady-state tissue concentrations may be very different, reflecting only its interaction with overlying water. A clam living in the sediment might experience little exposure because it filters overlying water that will never be in equilibrium with the sediment. Support for this comes from Foster et al. (1987), who found that a deposit-feeding clam (*Macoma balthica*) accumulated more PAH than a filtering feeding clam (*Mya arenaria*). To be complete in such analyses, potential metabolism of these compounds also needs to be considered to avoid confounding the conclusions.

Because most bioaccumulation toxicity tests and suhlethal toxicity tests can be several weeks in duration, adequate nutrition is essential for the health of the test species. Consequently, if the species is not a deposit feeder, some form of nutrition has to be added. For some toxicity test species, a food supplement is added (e.g., TetraMarin flakes), and in some long-term bioaccumulation toxicity tests with deposit feeders, new sediment is added periodically to ensure an adequate food supply. Additionally, some common toxicity test species (e.g., *R. abronius*) are predators, and standard testing protocols do not provide prey other than what may be resident in the sediment being tested.

The presence of fresh food, even for sediment-ingesting organisms, can affect the observed exposure. For the most part, bioaccumulation tests are performed in the absence of feeding, hut some require fresh food. For example, bioaccumulation of PAH congeners increased significantly (e.g., approximately by a factor of 2) with external feeding for both *H. azteca* and *C. riparius,* while the bioaccumulation of chlorinated compounds was not affected (Harkey, Landrum, Klaine 1994; Harkey et al. 1997). Thus, the addition of fresh food in laboratory toxicity tests can impact the observed bioaccumulation.

The results of bioaccumulation or toxicity studies in which additional food is required can be compromised if the food does not come into equilibrium with sediment and water concentrations. Individuals will ingest the added food, which may contain lower concentrations of the contaminant, and not the sediment or detritus that likely contain higher concentrations. For example, Bridges et al. (1997) examined the effect of food ration on a common toxicity test species, *Neanthes arenaceodentata.* The results of this study demonstrated strong effects on growth and survival as a function of the amount of food added to toxicity test chambers. Improved food quality and ration can lead to increased lipid, which in turn can increase bioaccumulation but also can provide storage tissue that can sequester lipophilic contaminants away from the site of toxic action. In addition, improved food quality can lead to increased growth, which will also reduce concentrations at the site of toxic action, hav-

ing a similar impact as added lipid. These results are particularly important fc the growth endpoint because variable food ration can be an important confounding factor when growth effects from toxicity are being determined.

Effect of contaminant interaction

When contaminants are present in low concentration, there does not appear to be any impact of 1 contaminant on the accumulation of others (Landrum 1989). However, there are cases in which the presence of certain compounds has acted to reduce the bioaccumulation of other contaminants. Specifically, the presence of polydimethylsiloxane (PDMS) was found to reduce the bioavailability of benzo[*a*]pyrene to *L. variegatus* (Kukkonen and Landrum 1995). This was the result of creating an additional sorption phase for the contaminant while the PDMS was not bioavailable. Even when a few compounds in a mixture are present in toxic concentrations, there is no evidence that these toxic compounds affect the relative accumulation of other compounds in the mixture (Landrum et al. 1989). Thus, when conditions are suc that a second sorptive phase is created in a sample, then bioavailability can be reduced. However, when all of the compounds are sorbed to the same matrix although some are producing toxicity, the relative accumulation does not appear to be affected. It should also he noted that the response to toxic compounds can affect the overall toxicokinetics.

Measurement artifacts

Artifacts in assessing bioaccumulation can arise in field or laboratory studies. Examination of Table 11-1 shows that some of the BSAF values for a given species (e.g., *Macoma* spp., for PAHs) can be highly variable (ranging from 0. to 1.3) across studies. This may be due to artifacts such as lack of gut purging field versus laboratory sources of contamination, static versus flow-through testing conditions, differing organism sizes or ages, differential contaminant bioavailability, and/or variable exposure time.

Performing bioaccumulation tests in the laboratory can be difficult and incor clusive if conditions found in the laboratory are not reflective of those in the field. Of course, many factors that vary in the field, such as temperature, pH, salinity, and oxygen content, are kept constant in the laboratory. Thus, the laboratory bioaccumulation test is a snapshot of one set of conditions found in the field. Other factors that are not often considered can be very importan to the outcome. For example, a bioaccumulation test can be static (without water changes) or flow-through (with water exchange at various turnover rates). The importance of this variable depends in part on the species and

chemical being tested. Some benthic invertebrates interact extensively with the water column and may derive a large proportion of their acquired tissue burden from filtering overlying water. However, for a deposit-feeding species such as *Diporeia*, the accumulation of PAH was the same whether the organisms were exposed under flow-through conditions or under static conditions (Landrum 1989). This was not the case for the amphipod *H. azteca*, where the observed toxicity was greater under static conditions than under flow-through conditions, reflecting differences in the accumulated toxicant, likely because of the epibenthic nature of the amphipod (Spehar et al. 1998). Additionally, the degree to which a chemical partitions between water and sediment may also be important. For very hydrophobic compounds, most of the accumulation will come from ingestion of sediment or prey (Landrum and Robbins 1990; Leppänen and Kukkonen 1998; Selck et al. 2003), so flow-though conditions may not affect the results.

Several important areas of research currently are aimed at enhancing our understanding of the nature of bioaccumulation. Studies on uptake efficiency from different sources, variability in digestive physiology among species, and qualitative and quantitative differences in organic carbon in determining bioavailability are but a few of the ongoing research projects leading the way to predicting both direct (tissue concentration–adverse effects relationships) and indirect (trophic transfer) effects more accurately and effectively in the future. Steps are also being taken to uncover the role of sediment particle size and relative contaminant distribution on bioavailability, the degree of equilibrium in field environments, the efficiency in trophic transfer of parent compounds and metabolites, and the intra- and interspecific toxicokinetic differences among compounds. Research in these and related areas will lead to enhancements in the current understanding of the processes governing contaminant accumulation and resulting effects in aquatic organisms.

Can Tissue Residue Concentrations Be Accurately Estimated using Theoretical Bioaccumulation Potential?

An EqP model, Theoretical Bioaccumulation Potential (TBP), has been used for nearly 2 decades as a screening tool to estimate the potential levels of bioaccumulation of persistent nonionic organic chemicals that could result in benthic organisms exposed to contaminated sediments. TBP has become a standard test in Tier II evaluations of dredged sediments proposed for open-water disposal (USEPA/USACE 1991, 1998; Chapter 6). A point estimate is

obtained by normalizing chemical concentration in sediments (C_s) on organic carbon content (f_{OC}) and by normalizing the expected concentration in an exposed organism's tissues (TBP) on lipid content (f_L) at steady state; the difference between the two is described by a coefficient, the BSAF:

$$TBP = BSAF \times (C_s / f_{OC}) \times f_L.$$

The empirically derived BSAF itself is calculated from normalized tissue and sediment data for the nonionic organic chemical of interest and is

$$BSAF = (C_t / f_L) / (Cs / f_{OC}),$$

where C_t is the measured tissue residue concentration. As risk assessment has gradually been introduced into the ecological sphere, it has become increasingly obvious that point estimates made with no associated measure of variability or uncertainty have limited utility. TBP is the simplest and most easily understood model for estimating bioaccumulation, but as such, it is also subject to a large degree of uncertainty. The model takes no account of the influences of kinetic processes that determine chemical bioavailability from sediments or retention, metabolic degradation, or elimination from organisms. These processes are integrated in the BSAF, which presumably will permit a good estimation of bioaccumulation, provided the conditions to which it is applied are similar to those from that it was derived. Although BSAFs are reported in the literature for organisms which have no direct association with sediments, the concept applies best to those that are benthically coupled, particularly sediment-processing infauna with relatively low xenobiotic metabolizing capability. This is because EqP derives from thermodynamics based on closed system in equilibrium. The concentrations of a chemical in contiguous environmental compartments of such a system are a function of chemical potential (or its ancillary, fugacity) and ideally are measured when kinetics are at steady state (i.e., when there is no further net exchange of chemical among th compartments). Therefore, whatever contributes to disequilibrium, such as metabolic degradation or distance from source to sink, necessarily contributes to uncertainty.

High-quality TBP estimations are necessary, first, because they are the simples and most easily understood means of estimating biotic exposure to sediment contamination and, second, because they represent the first step in any risk assessment involving sediment as a source. Thus, the quality of TBP estimation is highly dependent on the quality of selected BSAFs, whose variability may reach several orders of magnitude within a given classification.

Table 11-2 contains descriptive statistics calculated for BSAFs in a BSAF/lipid database (USACE/USEPA 2002b). The database (http://www.wes.army.mil/el/bsaf/bsaf.html) has been compiled from the open literature and from government reports and presently contains more than 1300 BSAF entries for organic chemicals. Lipid data for numerous species can also be found in the database. The BSAF data have been analyzed statistically by chemical class, specific chemical, laboratory or field exposure, organism habitat, and class of organism. Of course, the data may be grouped in any way desired, and there may be advantages to be gained, for example, by grouping by specific organism or specific chemical (or both). However, in the examples that follow, the statistics in line 6 of Table 11-2 were used to calculate uncertainty in TBP estimation for a benthically coupled fish exposed to PCB-contaminated sediments. In order to calculate uncertainty, replication in all input parameters is necessary.

Two methods for calculating the uncertainty associated with PAH TBP estimates have been suggested (Clarke and McFarland 2000) a root sum of squares (RSS) method and a statistical bootstrap method. The resulting uncertainty measures can be used in 3 ways:

1) to gauge the precision of TBP estimates,
2) to enable statistical tests of significance comparing TBP predictions with observed contaminant bioaccumulation, and
3) to provide inputs for risk assessments.

The RSS method computes total error from component method error and TBP data input propagated error. The bootstrap method uses computer-intensive resampling of the TBP data inputs to generate statistical uncertainty measures (e.g., standard error and confidence limits) and to perform tests of significance. Both methods are described in Clarke and McFarland (2000), and computational formulas are given for the RSS method. The methods are described in detail on the USACE/USEPA website in Technical Note EEDP-04-32 (http://www.wes.army.mil/el/dots/eedptn.html).

Recently, uncertainty analysis using the 2 methods was applied to estimating PCB bioaccumulation potential in a bottom-feeding freshwater catfish, *Ameiurus melas*, inhabiting a confined disposal facility (CDF) in Calumet Harbor, MI. The CDF covers 43 acres and is lined with a synthetic membrane of low permeability. Dredged material that has been disposed in the CDF is primarily contaminated with low levels of PCBs. Because there is virtually no advection of water through the system, it was considered that fish inhabiting the CDF pond would essentially be at steady state with the sediments,

Table 11-2 Descriptive statistics for BSAFs from the USACE/USEPA BSAF database [a]

Description	BSAF mean	*n*	SD	SE	CI of mean	Range	Max	Min	Median	25th percentile	75th percentile
1) All BSAFs in database	1.39	1179	2.78	0.08	0.16	41.47	41.47	0.001	0.48	0.10	1.57
2) All field studies	1.67	737	3.35	0.12	0.24	41.47	41.47	0.001	0.44	0.09	2.00
3) All lab studies	0.94	442	1.28	0.06	0.12	13.34	13.34	0.002	0.52	0.15	1.17
4) All benth[b] field	1.27	642	2.59	0.10	0.20	41.47	41.47	0.001	0.31	0.08	1.48
5) All benth[b] lab	0.97	428	1.29	0.06	0.12	13.34	13.34	0.003	0.53	0.18	1.29
6) All PCB benth[b] field	1.70	293	2.10	0.12	0.24	10.99	11.00	0.007	0.79	0.13	3.03
7) All PCB benth[b] lab	1.18	206	0.97	0.07	0.13	4.70	4.74	0.040	0.80	0.50	1.63
8) All PCDD/F invert[c] field	0.37	103	0.31	0.03	0.06	1.44	1.46	0.020	0.29	0.15	0.50
9) All PCDD/F benth[b] lab	0.24	30	0.23	0.04	0.09	0.90	0.90	0.003	0.17	0.13	0.27
10) All PAH invert[c] field	0.36	161	1.00	0.08	0.16	8.80	8.80	0.001	0.07	0.01	0.30
11) All PAH benth[b] lab	0.61	154	1.51	0.12	0.24	13.34	13.34	0.003	0.13	0.03	0.48
12) All pest benth[b] field	2.59	83	5.41	0.59	1.18	41.47	41.47	0.004	1.00	0.22	2.56
13) All pest invert[c] lab	1.91	31	1.74	0.31	0.64	5.49	5.88	0.390	0.80	0.58	3.88

[a] http://www.wes.army.mil/el/bsaf/bsaf.html
[b] benth = benthically coupled and refers to invertebrates and bottom-feeding fish, whole organism, muscle, or filet
[c] invert = invertebrates and refers to mollusks and polychaetes

in terms of contaminants. Approximately 30 sediment core composites were taken for PCBs as total PCB, Aroclors, and specific congeners and TOC. A similar number of fish samples were taken by electroshock for the same PCB analytes and for lipids. RSS uncertainty (Table 11-3) was calculated using the mean, n, and SE of line 6, Table 11-1 as the BSAF input data. Average method error (ME), the error inherent in the model equation itself if all measurements could be made with perfect accuracy, was found to be 49%. This value is similar to the average ME previously determined for PAHs (Clarke and McFarland 2000). Propagated error was evaluated separately and total error (TE) was calculated as the square root of the sum of the squared ME and the propagated error. The measured tissue concentration was found to lie within the limits of TBP ± TE 69% of the time and to overestimate TBP ± TE the remainder of the time.

The RSS method is relatively simple to perform and gives an estimation of the uncertainty surrounding estimations of bioaccumulation potential, but the bootstrap method has the advantage of calculating statistical measures, including confidence limits and probability. Using the bootstrap method on the same data set (Table 11-4), TBP overestimated the measured tissue concentration 6 times, the confidence interval contained the measured concentration 8 times, and TBP overestimated the mean measured concentration twice. However, when a test of significance was done on the analysis, there was only one significant overestimation ($P = 0.03800$) and 1 significant underestimation ($P = 0.03588$). All other comparisons found no significant difference between TBP and Ct.

What Other Methods are Available for Assessing Contaminant Bioavailability?

Semipermeable membrane devices and solid-phase microextractions fibers

Semipermeable membrane devices (SPMDs) and solid-phase microextraction fibers (SPMEs) have received considerable attention as useful tools for measuring organic compounds in environmental matrices. Both types of samplers are commonly referred to as "biomimetic devices" because they can be used as surrogates for organisms to estimate the bioavailability of organic contaminants. With regard to contaminant uptake, biomimetic devices share 2 features in common with organisms: 1) They selectively absorb "available" compounds, and 2) they can concentrate them to very high levels in comparison

Table 11-3 Computational (RSS) uncertainty analysis of PCB bioaccumulation potential in freshwater catfish *A. melas*

PCB	Mean BSAF[a]	Cs mean[b]	Cs CI[c]	TBP[d]	Propagated error	Total error	Range of total error		Ct mean[f]	TBP/Ct	Ct within range of error?
							TBP – TE[e]	TBP + TE			
Total	1.7	0.175	0.070	0.247	0.112	0.165	0.082	0.412	0.413	0.6	above
A1254	1.7	0.150	0.056	0.211	0.092	0.138	0.073	0.350	0.238	0.9	within
A1260	1.7	0.036	0.020	0.048	0.027	0.036	0.012	0.084	0.159	0.3	above
44	1.7	0.004	0.004	0.005	0.004	0.005	0.000	0.010	0.010	0.6	within
52	1.7	0.005	0.005	0.007	0.006	0.007	-0.000	0.014	0.011	0.6	within
77	1.7	0.004	0.004	0.006	0.004	0.005	0.001	0.011	0.009	0.7	within
86	1.7	0.003	0.001	0.004	0.002	0.002	0.001	0.006	0.002	2.4	within
101	1.7	0.005	0.003	0.007	0.004	0.005	0.001	0.012	0.013	0.5	above
128	1.7	0.002	0.001	0.003	0.001	0.002	0.001	0.005	0.003	1.2	within
138	1.7	0.010	0.004	0.013	0.006	0.009	0.004	0.022	0.006	2.3	within
141	1.7	0.002	0.001	0.003	0.001	0.002	0.001	0.005	0.003	1.0	within
156	1.7	0.002	0.000	0.002	0.001	0.001	0.001	0.004	0.004	·0.6	within
170	1.7	0.002	0.001	0.003	0.001	0.002	0.001	0.006	0.002	1.4	within
171	1.7	0.002	0.000	0.002	0.001	0.001	0.001	0.004	0.002	1.2	within
180	1.7	0.003	0.002	0.004	0.002	0.003	0.001	0.007	0.010	0.4	within

[a] BSAF from Table 11-2
[b] Concentration in sediment, ppm.
[c] 95% confidence interval.
[d] Theoretical bioaccumulation potential, ppm

Table 11-4 Bootstrap uncertainty analysis of PCB bioaccumulation in the freshwater catfish A. melas

PCB	Bootstrap mean TBP	Bootstrap std. error	Bootstrap CV	DF for CL[a]	Lower 95% CL	Upper 95% CL	Ct mean[b]	Ct within confidence limits?
Total	0.248	0.063	0.765	8	0.131	0.366	0.413	above
A1254	0.213	0.052	0.737	8	0.116	0.311	0.238	within
A1260	0.049	0.015	0.905	8	0.021	0.076	0.159	above
44	0.005	0.002	1.354	8	0.001	0.010	0.010	within
52	0.007	0.004	1.538	8	0.000	0.014	0.011	within
77	0.006	0.003	1.234	8	0.001	0.011	0.009	within
86	0.004	0.001	0.701	8	0.002	0.006	0.002	within
101	0.007	0.002	1.080	8	0.002	0.011	0.013	above
128	0.003	0.001	0.724	8	0.002	0.005	0.003	within
138	0.013	0.004	0.791	8	0.007	0.020	0.006	below
141	0.003	0.001	0.725	8	0.002	0.004	0.003	within
156	0.002	0.000	0.580	8	0.002	0.003	0.004	above
170	0.003	0.001	0.644	8	0.002	0.005	0.002	within
171	0.002	0.000	0.528	8	0.002	0.003	0.002	within
180	0.004	0.001	0.944	8	0.002	0.006	0.010	above

[a] DF for input parameter having smallest sample size (lipid, $n = 9$)
[b] Measured concentration in tissue, ppm (fresh weight)

to the surrounding matrix. In contrast to vigorous strong-solvent extractions, which attempt to sample 100% of the compound present, biomimetic devices sample only what is dissolved in solution or, in some cases, easily disassociated from other matter. Much like the organisms they attempt to mimic, SPMDs and SPMEs have large affinities for organic compounds, concentrating them to several orders of magnitude higher than the surrounding matrix. Similar to restrictions placed upon experiments using organisms to measure bioavailable contaminants, device–matrix volume ratios must be large to ensure the device does not deplete the "available" pool of compounds present in the matrix, and uptake should be measured preferably after reaching steady-state equilibrium.

The primary driving force behind the development of biomimetic devices is to provide a better estimate of exposure than is gleaned from vigorous strong-solvent extractions, while complimenting the use of live organisms for evaluating contamination in soils, sediments, and waters. Biomimetic devices are not substitutes for organisms but can provide complementary or preliminary information that allows a more efficient use of biota and a more refined understanding of toxicant exposure. Several key characteristics of biomimetic devices enable them to be easily integrated with an exposure assessment scheme. They are generally less expensive than using laboratory-reared organisms with a known exposure history. In some cases, exposure times are less than required for laboratory-reared organisms (deployed caged in situ or in laboratory toxicity tests). SPMEs and SPMDs can be deployed in most situations without regard to the myriad noncontaminant issues associated with organism needs for a matrix, such as nutrient or food quality, physical structure, and physicochemical conditions (pH, oxygen, light, etc.). Organisms caged in the field may die and/or be subject to predation. Contaminant uptake by an organism is influenced by a suite of variables, including genetics, age, behavior, handling, and overall health, each of which may contribute inherent variability compared to that of biomimetic devices.

Biomimetic devices are subject to a number of general disadvantages for measuring bioavailable organic compounds, in addition to device-specific disadvantages. The primary criticism of biomimetic devices is that they are overly simplistic (Salazar and Salazar 2001). SPMDs and SPMEs only simulate the dermal or respiratory (gills) route of exposure. For organics with log $K_{ow} > 5$, dietary routes of exposure become increasingly important (Leppännen and Kukkonen 1998); biomimetic devices may underestimate the bioavailability of such compounds. Biomimetic devices cannot account for biomagnification of persistent (usually very high log K_{ow}) compounds across trophic levels. Furthermore, biomimetic devices are simple, 1-compartment sinks for organic

compounds. They cannot simulate the complex compartmentalization of organics among different tissues, organs, and/or biomolecules, nor can they mimic biotransformation that results from metabolic reactions with biomolecules or detoxification systems (e.g., P450). Although these devices can lose accumulated molecules if external activity decreases, they cannot actively excrete compounds.

Though both SPMDs and SPMEs passively sample organic compounds, their designs are quite different. SPMDs are designed as high-capacity samplers and are usually used to detect trace or ultra-trace levels of compounds in air or water. SPMDs are layflat, thin-walled bags or tubes (standard size 106 × 2.5 cm) comprised of low-density polyethylene (LDPE) containing a thin film of the nonionic lipid triolein (standard volume 1 mL). Organic compounds that cross the LDPE membrane are sequestered in the triolein, mimicking storage of organics in fat. After exposure, the SPMD is subjected to a somewhat lengthy (1 to 2 d) procedure involving dialysis, size-exclusion chromatography, and fractionation or preconcentration before analysis via traditional gas chromatography (GC), gas chromatography–mass spectrometry (GC–MS), or high-performance liquid cbromatography (HPLC) methods (Petty et al. 2000). Although smaller-sized SPMDs can be used in laboratory situations (Wells and Lanno 2000), standard-sized SPMDs sample large volumes (e.g., 3 L/d for phenanthrene in water) and generally need a longer time period to achieve steady state, making in situ deployment the norm (Petty et al. 2000). Because of their long exposure times, in many environmental matrices, the LDPE membrane can become fouled with silt, periphyton, or bacteria, thus decreasing sampling rate (Petty et al. 2000). SPMDs usually are used to measure contaminant availability at the sediment–water interface (Echols et al. 2000; Petty et al. 2000; Voie et al. 2002). However, Leppänen and Kukkonen (2000) were able to bury small (2.7 × 2.5 cm) SPMDs in sediments spiked with pyrene and benzo[*a*]pyrene. Although neither worms nor SPMDs achieved steady state during the short (12 h) exposure periods, SPMD uptake of these compounds was well related to nonfeeding organism uptake and depicted a general decrease in contaminant availability in the sediments over time (Figure 11-1). However, SPMDs were not as effective in predicting uptake of organisms that were actively feeding (Leppänen and Kukkonen 1995). The intestinal route of uptake was found to be a significant exposure route, and therefore SPMDs underestimated bioavailability of the compounds to these organisms. Though SPMDs were able to mimic dermal or body wall exposure routes, caution should be exercised when using SPMDs if dietary exposure routes could be significant.

SPMEs are thin silica fibers (about 110 μm diameter, 1 cm long) coated with a microlayer (5 to 100 μm thick) of organic polymer. Unlike the triolein lipid found in SPMDs, the absorptive phase of the SPME is the polymer coating. Seven polymer coatings are available, ranging in analyte selectivity from polydimethylsiloxane (PDMS), useful for measuring very hydrophobic organics, to polyacrylate (PA), useful for measuring more hydrophilic organics. In contrast to SPMDs, SPMEs were developed for laboratory use in a wide variety of analytical applications, with their primary advantage being a reduction in solvent usage and freedom from chromatographic interferences (Pawliszyn 1997). SPME fibers are traditionally used mounted on a syringe applicator, enabling one to immerse the fiber in solution or in the headspace above a solution, retract the fiber, and directly inject the fiber into a custom GC or HPLC interface. In most cases, sample extraction, preconcentration, and purification are achieved in 1 step: No solvent is used, no cleanup is necessary, and the fiber may be reused. Although the syringe applicator method is very fast (steady state reached in minutes to hours in an agitated solution or slurry) and convenient, its use is primarily restricted to the laboratory bench top. With regard to sediments and soils, this application has been promising for predicting bioavailability in stirred pore water, overlying

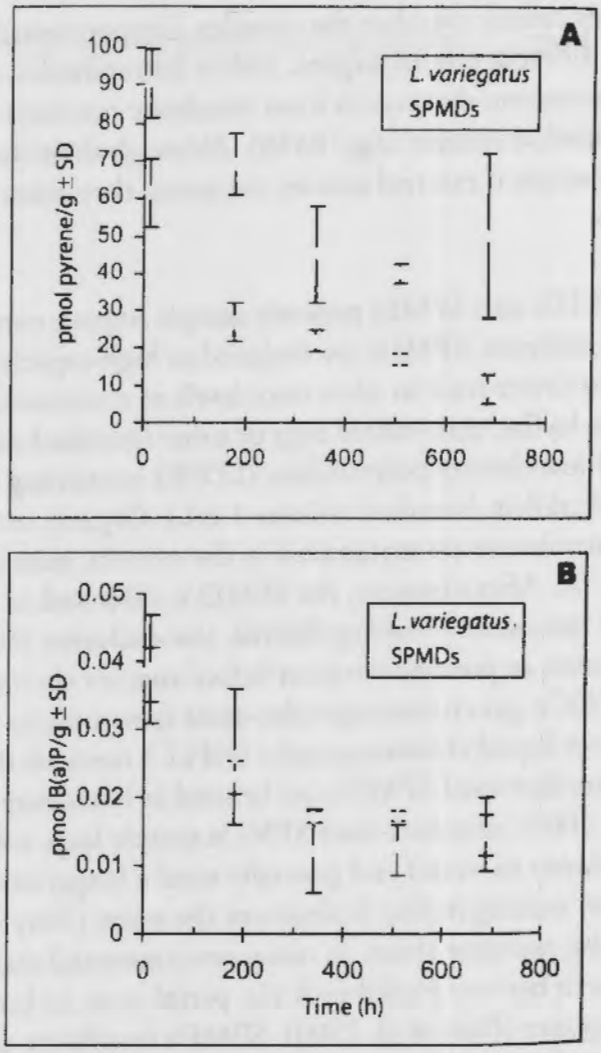

Figure 11-1 Accumulation of 14C-pyrene (A) and 3H-benzo[*a*]pyrene (B) in spiked Lake Höytiäinen (Finland) sediment to semipermeable membrane devices (SPMDs) and to nonfeeding *Lumbriculus variegatus* after five 12-h exposures in relation to sediment–chemical contact time. (Reprinted from *Aquatic Toxicology* 49:227–241, Leppänen and Kukkonen, Effect of sediment-chemical contact time..., copyright 2000, with permission from Elsevier.)

water, and slurries (Urrestarazu Ramos et al. 1998; Leslie et al. 2000, 2002; Wells and Lanno 2001). In a water-only exposure to *Chironomus riparius*, Leslie et al. (2002) derived 96-h LC50s based on water, organism, and SPME concentrations for a polar (2,4,5-trichloroaniline, TCA) and a nonpolar (1,2,3,4-tetrachlorobenzene, TeCB) narcotic compound (Table 11-5). A 3- to 4-fold difference was found for LC50s based on organism concentrations (CBRs). Assuming that the dose at the site of toxic action (cell membrane for narcosis) is similar for both compounds, the authors hypothesized that this large discrepancy in CBRs may be due to a higher toxicological bioavailability for the more polar TCA. That is, because polar organics tend to accumulate in the membrane, a lower CBR for TCA is to be expected. By comparison, LC50s based on SPME uprake for both chemicals were not only less variable than CBRs, they were nearly identical. Binding to this SPME coating (PA) may represent partitioning to cell membranes, rather than ro the whole organism (Vaes et al. 1997, 1998; Verbruggen et al. 2000). In this case, SPMEs provided a more precise, possibly more accurate estimate of the dose at the site of toxic action than did whole-body residues, which misrepresented the total exposure.

Table 11-5 Median-lethal doses (LC50), with 95% confidence limits, calculated using concentrations in water, organisms, or polyacrylate (PA)-coated solid-phase microextraction fibers (SPMEs) for *Chironomus riparius* exposed for 96 h in water spiked with 2,4,5-trichloroaniline (TCA) or 1,2,3,4-tetrachlorobenzene (TeCB)[a]

	LC50	
Dose metric	TCA	TeCB
[Water] (μM)	8.87 (7.31–10.8)	2.30 (2.13–2.49)
[Organism] (mmol/kg lipid)	38.7 (29.1–51.6)	144.2 (109.7–189.4)
[SPME] (mmol/L PA)	32.3 (29.4–35.6)	30.5 (29.7–31.3)

[a] Reprinted with permission from Leslie et al. 2002; copyright Society of Environmental Toxicology and Chemistry (SETAC)

Although there are published roxicological studies with SPMEs, all have been limited to the overlying water or water-only exposures of sediment. Slower static exposures via direct inserrion of the fiber into whole-sediment samples have been atrempred (Mayer et al. 2000). Because of rhe fiber's fragility, this technique was found to be problematic. Researchers at the University of North Texas (Conder et al. 2003) have developed a method to bury SPME fiber pieces (not mounted on syringe, cut to custom length) within steel mesh envelopes to measure compounds directly within the sediment of a toxicity

test without damaging the fiber. Currently underway is the development of a method to bury these envelopes within sediment in situ. Exposure times for static SPME are much longer than for traditional stirred SPME, taking 2 d for nitroaromatics sampled with PA-coated SPME to reach steady state and longer for more hydrophobic compounds.

SPME fibers have a few advantages over SPMDs for measuring available sediment-associated organic compounds, including costs and ease of use. Syringe-mounted SPME fibers are reusable and avoid costly solvent usage and disposal. For nitroaromatics, the mesh envelope burial method was found to be even less expensive than a traditional liquid–liquid solvent extraction to determine "total" organics. SPMEs need much smaller amounts of sample and achieve steady-state equilibrium much more rapidly than SPMDs. In the only published study comparing biomimetic SPMD and SPME, SPMD uptake was found to be roughly an order of magnitude higher than SPME uptake when phenanthrene was sampled from artificial soil (Wells and Lanno 2001). This increase in detection limit requires a larger sample size (i.e., 200 g for SPMD v. 1 to 2 g for SPME). However, because both the triolein in SPMDs and most SPME fiber coatings have extremely large affinities for organic compounds, both method detection limits sink well below levels producing toxicological effects for most toxicants. In some cases, sampling a small amount of sediment with SPMEs may be a disadvantage, particularly for heterogeneously distributed contaminants. In such cases, SPMDs may be ahle to provide a more robust integration of compound within the sample.

Dependent on study objectives, the use of biomimetic SPMD and SPME devices to estimate the bioavailability of sediment-associated organic compounds can provide a useful surrogate measure of contaminant availability. In general, biomimetic devices are easier to use, less expensive, less variable, and involve simpler chemical analysis, compared to measuring organic concentrations in organisms (Table 11-6). While SPMD and SPMEs do have limitations in predicting toxicity, the preliminary studies conducted to date have demonstrated their merit in investigating contaminant availability. SPMDs and SPMEs deserve more attention as complementary investigative tools for assessing the bioavailability of organics in sediment.

Deposit-feeder gut fluid extraction of sediment-associated contaminants

Deposit-feeding and some suspension-feeding organisms accumulate many heavy metals and hydrophobic organic compounds via the ingestion of sediment (Landrum and Robbins 1989; Wang and Fisher 1999; Lee et al. 2000;

Table 11-6 Comparison of organisms, semipermeable membrane devices (SPMDs), and solid phase micro-extraction fibers (SPMEs) for measuring the bioavailability of sediment-associated organic compounds

Sampler	Expense	Correlation with toxicity	Sampling time	Ease of use and analysis	Variability	Multiple exposure routes
Organisms	Higher	Better	Days to weeks[a]	More difficult or complex	More variable	Yes (ingestion, absorption)
			Hours[b]			
SPMDs	Lower	Good	Hours to weeks	Less difficult or complex	Less variable	No (only absorption)
SPMEs	Very low	Good	Minutes to days	Less difficult or complex	Less variable	No (only absorption)

[a] Laboratory-reared organisms
[b] Site-collected organisms

Weston et al. 2000). However, a substantial, often major, proportion of any given contaminant is not desorbed from the particles while in the gut and passes out of the organism via the feces. Environmental management decisions pertaining to contaminated sediments must include consideration of the bioavailable fraction rather than the total contaminant concentration. Chemical approaches to toxicant exposure have some advantages over biological ones, but existing chemical methods of analysis generally extract all of the targeted contaminant from sediments by using a strong acid or strong organic solvent. As a result, these approaches can overestimate the bioavailable fraction, by varying degrees, and can lead to misleading interpretations or noisy data.

Digestive fluids of benthic deposit-feeding organisms have been used as an extraction medium to provide a better assessment of the potential bioavailability of particle-associated contaminants (Mayer et al. 1996). Several investigators have attempted to improve human health risk assessment by developing fluids that mimic human stomach fluid and to use these fluids as in vitro extractants to estimate how much contaminant would be bioavailable from soil if incidentally ingested by humans (Ruby et al. 1993; Hack and Selenka 1996; Jin et al. 1999; Oomen et al. 2000). In the new method, digestive fluid of a deposit-feeding organism is removed from the gut lumen, and the sediments of concern are incubated with that fluid in vitro. The amount of the particle-associated contaminant that is desorbed in the fluid is then quantified on the presumption that sediment-associated contaminants must first be solubilized in order to be bioavailable (excluding the potential for intracellular digestion

in some taxa). While the approach does not address the subsequent absorpti of the solubilized contaminant across the gut wall, the method at least place an upper limit on the contaminant that is likely to be made bioavailable fror a given sediment during gut passage. The approach has the simplicity of a chemical extraction, but by using digestive fluid rather than an exotic solven the approach may provide more biological realism than is achieved by conve tional chemical methods. The digestive fluid extraction approach is probably not useful for compounds for which ingestion is likely to be a minor route o uptake (e.g., hydrophilic organic compounds; Weston and Mayer 1998) or f contaminants in which intestinal absorption rather than solubilization constrains uptake (e.g., Cr).

Recent attempts to assess sediment risk using in vitro digestive fluid extractic have illustrated some advantages of the approach over conventional measures of bioavailability involving exposure of live organisms (Weston and Maruya 2002). First, it can be done much faster than conventional bioaccumulation testing (a few hours versus nearly a month), with associated cost savings and faster data availability. Second, the digestive fluid approach to predict bioavai ability eliminates the potential effects of biotransformation. A comparison of results from digestive fluid extraction to measured tissue concentrations can help to elucidate the role and importance of biotransformation in a particula organism. Third, the technique allows evaluation of sediments by a consisten method over a wider range of abiotic parameters (e.g., grain size, salinity) tha would be tolerated by any single bioaccumulation test species. While reliance upon natural populations of deposit feeders as a source of digestive fluid limit widespread use of the approach, the use of commercially available substances having extraction properties similar to the natural constituents shows promise (Chen and Mayer 1999; Ahrens et al. 2001).

It is theoretically defensible to suggest that in vitro gut-fluid contaminant extraction might provide a direct measurement of contaminant bioavailability to deposit feeders. In addition, the method may be useful as a predictive tool for contaminant bioaccumulation in sediment-dwelling invertebrates, dependent upon certain conditions. To illustrate this extrapolation, one must first consider the steps whereby a contaminant is released from sediment and accumulated ultimately within the tissues of a benthic organism. The concentrations or fluxes of sediment-associated contaminants bioaccumulated via ingestion by deposit feeders can be considered using a multi-step model (Figure 11-2). The first 2 functions (a and b, Figure 11-2) consider the amount of contaminants transferred from the sediment into the gut fluids, calculated as the amount of contaminant solubilized by the gut fluid (a) minus the amount

subsequently reabsorbed into the sediment matrix (b). The difference (a – b) equates to the net in vitro gut-fluid extracted contaminant. The amount of contaminant absorbed from gut fluids into tissues (c, Figure 11-2) is proportional to the absorption efficiency. Final tissue burdens are a product of the absorbed fraction minus losses due to metabolism and elimination (d, Figure 11-2). Using this construct, it is evident that bioaccumulation is proportional to, and therefore theoretically predictable by, bioavailability. However, if a contaminant is rapidly biotransformed, digestive fluid extraction may predict high bioavailability, and the compound may indeed be taken up quite readily, but biotransformation of the compound could result in little or none of the substance being measured in the tissues. Bioaccumulation at steady state should be a correlate of in vitro solubilization for substances that are not biotransformed (e.g., DDE), for taxa having poor biotransformation capabilities (e.g., bivalves), or when values for biotransformation can be empirically estimated. Regression analyses of gut-fluid extracted contaminant concentrations versus bioaccumulated body burdens reveal strong positive correlations for a number of contaminants, suggesting that gut-fluid extractions might be considered as predictors of bioaccumulation (Lawrence et al. 1999; Weston and Maruya 2002).

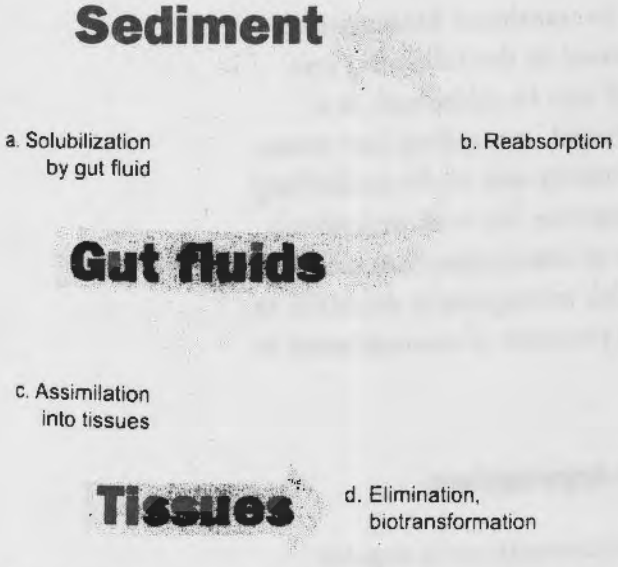

Figure 11-2 Conceptual model showing bioaccumulation of contaminants from sediments via deposit feeding

Can Bioaccumulation at Higher Trophic Levels Be Predicted Using Current Modeling Techniques?

The preceding sections have generally dealt with factors affecting bioaccumulation at lower trophic levels, such as polychaete worms or mollusks. This is important because in most ecosystems, the overall biomass of organisms at lower trophic levels is greater than the biomass at higher trophic levels (Valiela

1995). Thus, initial bioaccumulation of contaminants at lower trophic levels generally represents the major route of entry for contaminants into biotic systems. To understand the effects of contaminants on overall ecosystem form and function, however, one must also understand the efficiency with which contaminants that initially accumulated at lower trophic levels are transferred to higher trophic levels. Whereas contaminant uptake modeling at lower trophic levels focuses primarily on contaminant bioconcentration from the surrounding media (sediment particles, pore water, overlying water, etc.), biomagnification modeling at higher trophic levels attempts to more explicitly consider food as the primary route of exposure. Trophic transfer models range from kinetic and equilibrium modeling at the individual level to whole ecosystem approaches such as radiolabeling in order to assess trophic structure and subsequent potential for contaminant biomagnification.

The models that currently are used to estimate contaminant biomagnification at higher trophic levels will be briefly discussed in the following text. Uncertainties associated with each approach will also be addressed. It is important to note that although parameter or model uncertainty can potentially be reduced with additional research, uncertainty due to the underlying inherent variability in uptake or contaminant response between individuals or between species cannot be reduced. This type of uncertainty can only be quantified, understood, and incorporated into risk management decisions in determining the acceptability of risks due to the presence of contaminants in sediment.

Individual organism: Equilibrium-based approaches

Although field studies show increases in tissue concentrations in organic contaminants at higher trophic levels (e.g., Oliver and Niimi 1988), animals higher up on the food chain also tend to have greater lipid content than organisms in lower trophic levels (LeBlanc 1995). In fact, for some organic contaminants, lipid-normalized concentrations do not vary widely at different trophic levels (LeBlanc 1995), indicating that although food can be a major route of exposure, aquatic organisms may remain in simple equilibrium with surrounding media. Alternatively, while contaminant concentration in ingested food may be high, exposure from other routes may be more important. For example, although contaminant concentration in water or suspended particles may be low, the large volume of water processed by filter feeders or passed through the gills of fish may lead to these media being the dominant pathways of exposure.

If the contaminant assimilation efficiency is inversely related to the organic carbon content of the water or particles, and the elimination rate is inversely related to the lipid content of the organism (LeBlanc 1995), then at steady state the lipid-normalized tissue concentration of organic contaminants will be related to the organic carbon–normalized contaminant concentration in the surrounding media. In both cases, the lipid-normalized concentration of organic contaminants may not vary significantly between different trophic levels and can be considered to be in equilibrium with the organic carbon–normalized concentration in the surrounding sediments or water. If this is the case, evidence for true biomagnification (food ingestion leading to an increase in tissue concentration over simple equilibrium bioconcentration) is not always observed. In a model of field data by LeBlanc (1995), evidence that biomagnification is due to food ingestion was observed only for compounds with a log $K_{ow} > 6.3$. If this is true, modeling of many contaminants at higher trophic levels can use the same equilibrium bioconcentration models used at lower trophic levels.

Individual organism: Kinetic approaches

The simplicity of equilibrium models makes them an attractive choice as predictive tools for determining bioaccumulation of organic contaminants at higher trophic levels. However, trace metals are rarely in equilibrium with aquatic sediments, either spatially or temporally. Interactions between inorganic sediment particles (comprised in part by both macrofauna and microbial populations) result in distinct concentration gradients and relatively rapid geochemical cycling of associated metals. Thus, within complex sedimentary systems, exposure of an organism to contaminant metals is determined by the concentration and bioavailability of the trace metals at a scale proximate to the organism. Therefore, depending on a specific animal's feeding and living habits, metal exposure to a benthic organism can be substantially different than that predicted by metal concentrations measured in bulk sediment or food alone. In this regard, equilibrium models applied to benthic ecosystems may be inappropriate.

Metals may be accumulated from both dissolved and dietary sources (Wang et al. 1996). Recent work has focused on developing a mechanistic understanding of metal uptake by separately evaluating the contribution of each potential source to the total metal accumulation in aquatic organisms. For site-specific exposure assessment, Luoma and Fisher (1997) defined a simple kinetic model approach that includes empirically derived species-specific rate constants of uptake and loss. Key parameters determined experimentally include metal

influx rates from solution, influx rates from ingestion, and efflux rates. These adjustable constants are combined with environmental data (metal concentrations in pore water, overlying water, surface sediment and/or suspended particulates) representative of a range of conditions in the system of interest. Kinetic modeling results so far appear to predict metal concentrations in the few species studied (Luoma et al. 1992; Wang et al. 1996, 1999; Roditi et al. 2000; Griscom et al. 2002) and further studies hopefully will find ways to improve the model.

For many aquatic invertebrates, ingested food accounts for a major proportion of total trace metal accumulation. For metals accumulated primarily from food, trophic transfer models are useful in quantifying the potential for the specific contaminant to be magnified or minimized from 1 trophic level to the next. The trophic transfer potential (TTP) can be defined by the equation:

$$IR \times AE / [k_e + g]$$

where IR is the weight-specific ingestion rate, AE is the assimilation efficiency of the metal from the food item, k_e is the physiological loss rate constant, and g is the weight-specific growth rate. Recent work devoted to the quantification of AE and k_e for a variety of organisms and metals has provided the data needed for comparative studies among different species (Reinfelder et al. 1998). Results suggest that Ag and Cr are not biomagnified; some elements, Se and Cd in particular, may be somewhat biomagnified in bivalves consuming phytoplankton; and methyl-mercury is highly biomagnified in all aquatic organisms studied.

Kinetic models have been used to predict trace element concentrations in aquatic organisms for more than 10 years. However, these methods are not yet incorporated into SQGs. Problems that are of concern regarding the viability of kinetic models as a predictive tool may include, for example, the large number of required parameters. Kinetic models are not one-size-fits-all, but instead are best at describing what is, in fact, a complex and variable system and are site-specific. In addition, variations in food sources can have large effects on the AE (Lee and Luoma 1998; Griscom et al. 2000), and altered ingestion rates can effect the loss rate k_e (Reinfelder et al. 1998). Both of these variables can change the relative importance of the route of uptake, and the loss rate term k_e may not be best described by a single value. For example, exposure of organisms to high metal concentrations can trigger the production of detoxification products (e.g., metallothioneins or phosphate granules), which depending on the amount of metal concentrated in each fraction, may require additional loss-rate terms to be added to the model. Nevertheless, aside from the aforementioned issues, the model is designed to be adjustable, and thus it

can be used not only to predict bioaccumulation but also to quantify the route of uptake.

The kinetic models described above treat the animal as a single compartment. However, 1-box kinetic models may not be appropriate for larger organisms such as fish or other organisms in which metals are subject to feedback among various internal compartments. In general, models that explicitly incorporate mechanistic observations will prove most useful in the long term, and advanced compartmental models (Thomann et al. 1997, for Cd in trout) represent an improvement in assessing trace element accumulation. Concerns regarding the extensive data requirements for kinetic models may be less of an issue as the importance of specific variables becomes better understood. Eventually, it may be possible to estimate some of the species-specific parameters for ecologically similar organisms as more modeling studies are completed and general patterns emerge.

Whole ecosystem approaches

Even when all the kinetic or equilibrium parameters are known for a given species, assessing biomagnification in higher trophic levels can prove difficult. In order to estimate biomagnification at higher trophic levels, one must know the steady-state (or equilibrium) concentration of contaminants at lower trophic-level organisms serving as the food source in addition to knowing the feeding strategy used by the higher trophic-level organisms. Historically, this has been done by attempting to assign organisms to discrete secondary consumer, or higher, trophic levels and applying trophic transfer factors as described in the preceding section. However, organisms can feed at different trophic levels, for example, feeding on both zooplankton and small planktivorous fish, and thus do not fall neatly into specific classes. In a study of the biomagnification of Hg in lake trout, the concentration of Hg in this top predator was highly variable between a number of lakes (Cabana and Rasmussen 1994). Food chain length, and thus trophic class and Hg concentration in food sources, varied among the lakes. Not surprisingly, Hg concentration in lake trout, the top predator, was higher in lakes with longer food chains. This result is similar to previous work (Rasmussen et al. 1990) for PCBs in Ontario lakes.

Stable nitrogen and carbon isotopes have been used as markers of trophic status in aquatic ecosystems (Pererson and Fry 1987). This is because the $\delta^{13}C$ or $\delta^{15}N$ of an organism is slightly enriched over the isotopic ratio in the food source (Peterson and Fry 1987). Therefore, organisms at higher trophic levels are enriched in the heavier isotope. Rather than constraining organisms to an

arbitrary trophic position based on assumptions of feeding behavior, the isotopic ratio serves as a continuous variable, integrating feeding behavior and thus trophic status. In the study by Cabana and Rasmussen (1994), although the lake trout Hg concentration was variable between lakes because of differences in food chain length, the Hg concentration was correlated with $\delta^{15}N$. That is, trophic status, and thus biomagnification potential, is reflected in the isotopic content of the trout. Nitrogen and carbon isotopic content have been used to explain the biomagnification of contaminants at higher trophic levels in both freshwater and marine systems (Jarman et al. 1996; Vander Zanden and Rasmussen 1996).

Although the concept of stable isotopic modeling of contaminants is sound, there are problems in its application. In a study on the concentration of Hg in yellow perch, the authors concluded that between-lake variability was better described by within-lake differences in chemistry, and not by differences in trophic position. In addition, the decidedly nonconservative behavior of nitrogen in marine ecosystems often complicates interpretation of isotopic data in higher organisms (Cloern et al. 2002). Finally, while the isotopic composition of higher organisms may "explain" the biomagnified concentration of contaminants, it is not possible to set up a priori models predicting trophic structure, biomagnification potential, and adverse outcomes at the ecosystem level. Food chain length and breadth are likely site specific, thus no overall model will apply universally to all ecosystems.

How Does One Determine Acceptability of Predicted or Measured Chemical Residues Using a Risk-Based Approach?

Knowledge of the biogeochemical factors that affect the partitioning and fate of contaminants in sediments is improving. As a result, a variety of models and measurement tools have been developed to evaluate the potential for toxic chemicals to bioaccumulate from sediment into aquatic organisms, including biomagnification along a food chain. However, bioaccumulation itself is not an adverse effect. Bioaccumulation becomes an adverse effect only when contaminants accumulate within an organism to levels that elicit an adverse response. Therefore, in order to incorporate bioaccumulation potential into the decision-making process, decision makers need to understand the extent to which any observed bioaccumulation in aquatic organisms will adversely affect ecological or human health.

In recent years, the field of risk assessment has been used to assess the likelihood that exposure to contaminants will result in unacceptable outcomes. This requires both knowledge of how organisms are exposed to contaminants and an understanding of the toxicological effects associated with that level of exposure. Risk assessments can be used retrospectively to determine whether unacceptable risks have resulted from previous exposures. This type of assessment is used, for example, to determine if current levels of contaminants in a sediment are of concern and thus merit remediation. In addition, risk assessments can be used to predict future risks on the basis of future exposure scenarios. This is the type of assessment that is used to set site-specific cleanup goals for a contaminated site or to develop more widely applicable SQGs predictive of risk (or lack thereof) to either human or ecological receptors.

The 3 basic phases of a risk assessment are

1) determining important receptors, endpoints, and exposure pathways of concern,
2) collecting and analyzing exposure and effects data, and
3) characterizing risk (USEPA 1998; Chapter 6).

The first phase, identifying endpoints of concern, entails defining valuable aspects of the environment considered to be at risk. Important questions in Phase 1 include, What species do we care about? How much do we care about them? How are they exposed to the contaminants? What contaminants do we care about? What models or measurements will we need? How will the data relate to these endpoints? Issues such as societal or biological relevance and accessibility to prediction or measurement are important. Failure to unambiguously address these issues up front often results in a program that fails to adequately characterize risk to either human or ecological receptors (Chapter 6). How bioaccumulation potential of sediment-bound contaminants is used to assess ecological risks, therefore, is primarily a function of how risk managers choose to define ecological health. In other words, it is impossible to determine how bioaccumulation potential provides answers without first figuring out what the important questions are. It is beyond the scope of this chapter to discuss in detail the types of questions and endpoints required of an ecological or human health risk assessment. A more detailed discussion of the design of risk assessment programs, especially ecological risk assessments, is given in Schmitt and Osenberg (1996) and Suter (1993).

The second part of a risk assessment, characterizing exposure, is what this chapter has discussed. With respect to exposure in ecological receptors, this chapter has reviewed the various models and approaches used to estimate the

uptake of toxic contaminants into organisms. As will be discussed, exposure estimates must be used to model and act upon estimated risks of unacceptable toxic outcomes. For human health risk assessments, the primary route of exposure to sediment-bound contaminants is through oral consumption of fish or shellfish that has bioaccumulated those contaminants, either through direct contact with the sediment or via biomagnification along a food chain. For these assessments, models need to address not only contaminant concentration in the food source but also consumption rates.

Finally, the last part of a risk assessment entails using the exposure data to characterize actual risk. This implies knowledge of dose–response relationships for each contaminant and each species of interest. With respect to bioaccumulation of contaminants into aquatic organisms, how tissue levels of contaminants correspond to doses associated with adverse effects needs to be understood, including the temporal aspects of the dose–response relationship (Lee et al. 2002). For human health, a relation to the ingestion rate of contaminants with the likelihood of adverse cancer and noncancer outcomes is needed. The following briefly describe current risk assessment methodologies for both ecological and human health outcomes. The goal is to demonstrate major uncertainties in the application of these risk assessment models.

Risk assessment for ecological health endpoints

An obvious concern with contaminated sediments is that the presence of contaminants does not cause unacceptable environmental degradation. Determining unambiguous measurement endpoints that assess "environmental degradation" is difficult. An advantage of ecological risk assessment over human health risk assessment is the fact that it is possible to test the effects of contaminants directly on a species of interest. However, unlike human health assessments, there are usually more than 1 species to protect. The degree of extrapolation from tests on a few indicator or standard test species to other components of the ecosystem is the subject of considerable debate (Suter, 1993). Similarly, extrapolation from the results of a few acute or chronic toxicity tests to overall ecosystem health is of concern. When levels of biological responses are considered, ranging from subcellular and cellular responses, through individual and population responses, to responses altering overall ecosystem form and function, using bioaccumulation to assess ecological risk is extrapolating from the level of individual organisms to assess ecosystem health. Some monitoring programs extrapolate all the way from biomarkers at the subcellular level (such as enzyme induction, gene expression, or lysosome integrity) to indicate overall ecosystem health (IOC 1996). While this may be excessive, it

may be possible to develop quantitative or qualitative subcellular biomarkers of individual health, such as biomarkers indicative of exposure and thus bioaccumulation, to aid in assessing overall ecosystem health. However, much more research is needed in this area before these approaches can readily be applied routinely in ecological risk assessments.

Assuming an ecological endpoint of interest is acute or chronic toxicity in native organisms, the CBR approach has been used to model dose–response relationships in aquatic organisms (McCarty and Mackay 1993). In classic mammalian toxicology, "dose" is often defined as an amount (or rate) of a chemical delivered to an organism, with units such as mg contaminant/kg of body weight/day. Adverse toxicological responses are estimated as a function of the magnitude of the dose. However, in ecotoxicology it is often difficult to measure dose in this way, especially in field studies. Commonly, the concentration of contaminants in the surrounding medium is used as a surrogate for dose in ecotoxicological studies (Rand et al. 1995). Toxicity, therefore, is measured as a function of concentration, not as a function of the classic definition of dose. The CBR approach requires an understanding of the critical dose to an organism that elicits an unacceptable adverse effect (measured as a concentration in the surrounding medium). Models discussed in this chapter can then be used to additionally estimate the body burden of contaminants at that critical dose. Essentially, the body burden of contaminants can be used as an estimate of adsorbed dose. If the biogeochemical factors controlling bioaccumulation are understood, the concentrations of contaminants leading to unacceptable responses can be determined.

A CBR type of approach has a number of strengths. Primaty strengths are that bioavailability, exposure via food, and accumulation and depuration rate kinetics can be explicitly addressed. More sophisticated modeling of contaminant uptake from the environment to estimate subsequent tissue levels indicative of adverse doses. In general, CBR models for nonionic organics tend to be more developed than CBR approaches for heavy metals. This is because the mode of action for these contaminants is a narcosis, or nonspecific effect on lipid membrane integrity (Escher and Schwarzenbach 2002). The bioaccumulation of nonionic organics into lipid tissue is somewhat easier to model than the bioaccumulation of metals, which can be more effectively regulated by the organism. However, although CBR approaches are generally used for organic contaminants, the approach can be used for heavy metals as well (McCarty and Mackay 1993; Shephard 1998; Hamilton 2002).

A major uncertainty in the use of the CBR approach is determining a dose or response protective of ecological health. A large amount of acute toxicity data

is on acute mortality. However, using an acutely toxic dose to calculate a corresponding threshold CBR may not be protective of ecological health. One approach circumventing this problem is to use USEPA water quality criteria (WQC) to estimate target CBRs. This is based on the assumption that WQC are protective of a large fraction of species present in the environment. By determining the bioconcentration of dissolved contaminants into soft tissue, one can estimate a body burden, or CBR, of contaminants corresponding to a biological dose presumed to be protective of ecosystem health (Shephard 1998). This CBR value can be subsequently applied to sediments, where multiple routes of exposure (water, particles, food) can be modeled to determine sediment concentrations that yield an anticipated body burden less than the CBR indicative of an unacceptable dose. In short, tissue levels of contaminants can be a very useful tool to measure dose from multiple exposure pathways and can be compared with CBR-based estimates of unacceptable doses. This is especially useful for sediments, for which there are often multiple routes of exposure. However, as plagues all ecological risk assessments, what one chooses to define as an unacceptable outcome protective of ecosystem health is very difficult to determine.

Establishing the tissue residue that is protective as suggested by Shephard (1998) or establishing a CBR that reflects a toxic response allows the potential for establishing an SQG through the use of the BSAF. The approach to establishing the body residue can use ambient WQC and an appropriate BCF to establish a protective body residue (Shephard 1998). Mixtures are then addressed in this approach by the use of a toxic units model that assumes additivity. Similarly, it is possible to establish the body residue at some level of toxic response by using the LC50 or EC50 or other appropriate level of response and the corresponding toxicokinetics. The simplest approach would be to assume toxicokinetics at steady state and then use the product of the LC50 and the BCF to yield a body residue corresponding to the LR50 (50% mortality based on tissue residue). If the species-specific toxicokinetics are known, then temporal variations in the body residue–response relationship could be addressed at other than steady state. However, steady state in the environment would likely dominate the expected exposure for most cases. Finally, the body residue could also be established through experimental measurements (note: this is becoming more common and data are available in published databases [e.g., Jarvinen and Ankley 1998; USACE/USEPA ERED 2002a]). All of these approaches to establishing the body residue–response relationship can be applied through the BSAF to relate the body residue to the concentration in the sediment, assuming steady state. The BSAF value needs to be applied based

on the value for the specific compound class (Table 11-2). Alternatively, site-specific BSAF values can be generated and applied to account for site-specific factors. To develop site-specific BSAF values, data should he obtained from several species from diverse taxa. A BSAF is then selected from the upper end of the distribution (e.g., 95th percentile) to protect the most sensitive species. It may be that a set of BSAF values will be developed for a site-specific target organism to insure the protection of that species. The BSAF values are then used in the following equation to establish a compound-specific sediment concentration:

$$C_s/f_{OC} = \frac{[\text{tissue}]}{\text{BSAF} \times f_L}$$

where [tissue] is the concentration (e.g., lethal residue [LR50], effective residue [ER50], lowest effect residue [LOER], or other tissue residue response value as suggested above), BSAF is the biota–sediment accumulation factor, f_L is the fraction of lipid in the organism, and C_s is the chemical concentration in sediment and f_{OC} is the fraction of organic carbon in the sediment. Because this sediment value is for a specific compound, mixtures would need to be addressed, again using a toxic unit approach. This then leads to the possibility of establishing bioaccumulation-based sediment concentrations protective of toxic responses for a wide range of endpoints, both acute and chronic.

Two recent examples of this approach can be found in Meador et al. (2002a, 2002b) . Several studies with sufficient data to determine the tissue concentration associated with effects of tributyltin exposure were used to calculate the LR50, a growth LOER, and a threshold residue for sterility in stenoglossan gastropods (Meador et al. 2002b). This information, coupled with BSAFs for several species, was then used to determine the sediment concentrations expected to produce the adverse effects. These sediment concentrations were generated as guideline values for use in assessing sites contaminated with this compound. A similar approach was used to determine sediment concentrations of total PCBs that would likely cause adverse effects in juvenile salmonids (Meador et al. 2002a).

Human health–based risk assessments

In addition to protecting ecological health, environmental decision makers must also protect human health. In contrast to ecological risk assessments, in a human health risk assessment there is only 1 species of interest. Furthermore, whereas an ecological risk assessment is often aimed at maintaining a stable population of a species, a human health assessment is generally trying to protect the health status of each individual. Although routes of exposure can

include dermal contact or direct sediment ingestion, human exposure to sediment-borne contaminants is generally via consumption of contaminated food from the aquatic environment. Assuming the biogeochemical factors controlling the bioaccumulation of contaminants from sediments into aquatic organisms are understood, a human health risk assessment focuses on using these data to relate contaminant intake via food consumption (dose) with the likelihood of adverse health outcomes (response). The 2 major aspects of a human health risk assessment, therefore, are estimating aquatic food consumption rates and understanding the potential for adverse effects associated with a given dose of contaminants.

A variety of approaches are used to estimate risks from consumption of contaminated seafood. A common approach relies on toxicity data summarized in the USEPA's Integrated Risk Information System (IRIS) Database, which provides data on doses of contaminants associated with risks of both cancer and noncancer outcomes (USEPA 2002). For noncancer outcomes, the IRIS database provides a threshold contaminant dose (mg contaminant/kg body wt/time), below which the likelihood of a specific noncancer outcome is acceptably low. For cancer outcomes, the IRIS database provides an estimate of the incremental increase in the likelihood of cancer associated with a given rate of exposure (e.g., the cancer slope factor). As valuable as these consensus dose–response factors are in estimating risk, they are the subject of considerable debate. Because direct testing of humans is considered unacceptable, much of the data on human susceptibility to contaminants are based on extrapolation from mouse or rat studies. Some toxicologists feel that rat or mouse extrapolations to humans are unreasonable, while others feel that there is sucb underlying interindividual variability in susceptibility to these toxicants as to render the fixed thresholds meaningless (Crouch 1983). Thus, our ability to reliably estimate human health risks from the bioaccumulation of sediment contaminanrs into seafood is already compromised by our inability to precisely predict the effects of these contaminants on humans, either because of imperfect extrapolations from mouse or rat studies or by large variability in interindividual responses to contaminant exposure.

Putting aside uncertainties with the USEPA's cancer slope factors or non-cancer reference doses, risks from seafood consumption are modeled as a function of contaminant ingestion rate. Contaminant ingestion rates, sometimes called a "lifetime average daily dose" (LADD) are derived as follows:

$$\text{LADD (mg/kg/d)} = (\text{CF} \times \text{IR} \times \text{ED}) / (\text{BW} \times \text{AT}),$$

where CF = concentration of the contaminant in fish (mg/kg wet weight), IR= fish ingestion rate (kg fish/d), ED = exposure duration (y), BW = body weight (kg), and AT = averaging time (y). Guidance on default values to use for ED, AT, and BW can be found in documents such as the USEPA Guidance Manual (1989). The CF term can be estimated using the tools described in this chapter, although there are certain caveats with respect to the portion of the fish consumed.

Deserving further discussion is the IR, which can be highly variable. One must understand the distribution of fish consumption rates in the population under consideration in order to adequately protect that population from adverse risks. Risk managers must decide on a consumption rate that is representative of an acceptably high proportion of that population. It is clear that risks will vary with specific subpopulations that have markedly different dietary intakes, for example, subsistence fishers. Variability in the IR term that results in interindividual variability in fish consumption rates can lead to imprecision of final risk estimates. However, this source of uncertainty is due to stochasticity, or natural variability, and cannot be reduced through further research on fish consumption patterns. Uncertainty in the IR term must be understood and used in determining a target IR rate for the exposure models. Alternatively, probabilistic Monte Carlo models can be used to propagate this uncertainty and determine a distribution (rather than a point estimate) of exposure doses to the population of interest.

Once an LADD is calculated, one determines an excess cancer probability from that exposure by multiplying the LADD by the cancer slope factor. For noncancer outcomes, the LADD is divided by the "safe" reference dose to get a hazard quotient. As with ecological risk assessment, before one can back-calculate a sediment concentration based on a target risk level or calculate risks due to current conditions, an acceptable level of risk is established. A cancer probability less than 10^{-6} is generally considered to be acceptable, while a probability greater than 10^{-4} is generally thought to be unacceptable. Selection of acceptable criteria between these 2 extremes is a subjective risk management issue. Given that what can be considered an acceptable cancer risk can vary by a factor of 10 to 100, allowable amounts of contaminants in sediments can vary widely based on the risk threshold selected. For non-cancer outcomes, a hazard quotient less than 1.0 is considered acceptable. When values are greater than 1.0, establishing at what level risks become unacceptable is a risk management decision. As an example, the state of Washington has a program incorporating human health risk based criteria into setting cleanup goals for contaminated sediments (Washington Department of Ecology

1997). Although the target cancer risk threshold is 10^{-5}, consideration of other criteria, such as background concentrations of contaminants, has led tc cases where the estimated cancer risks associated with final cleanup goals are higher than the desired 10^{-5} risk threshold (Washington DOE 1997).

A second type of threshold used to estimate human health risks that result from food consumption in aquatic environments: comparison to USFDA action levels of allowable amounts of contaminants in food. The USFDA has published a list of allowable amounts of contaminants in food and seafood (USFDA 2002). One can therefore consider sediment concentrations unacceptable for humans if they result in fish tissue levels above the USFDA action levels. However, it should be pointed out that USFDA action levels are not solely based on estimates of human health risk. Economic factors (e.g., availability of alternative food sources) are also incorporated into the establish ment of the action levels (21 CFR 109; 21 CFR 509). In certain instances, th health risks associated with a USFDA action level may be higher than desired For example, a recent report by the National Academy of Sciences investigatec human health risks associated with the consumption of Hg-contaminated sea· food (NRC 2000). The report indicated that the USFDA action level of 1.0 μg/g may not be protective of human health and recommended lower tolerance levels for Hg in fish tissue (NRC 2000).

With respect to the exposure equation given above, another source of uncertainty is the fish concentration of contaminants that should be input into the exposure model. Much of the discussion in this chapter has described bioaccumulation of contaminants into the whole body of organisms. In many instances, however, contaminants are not distributed uniformly throughout the body. For example, in the American lobster (*Homarus americanus*), the body burden of bioaccumulated organic and inorganic contaminants is often concentrated in the hepatopancreas and these tissues may or may not be consumed by humans, depending on individual or cultural preference (Canli and Furness 1993; James et al. 1995). In fish such as salmon, bluefish, and carp, it has been shown that the concentration of nonionic organics in skin is higher than in muscle tissue (Hora 1981; Armbruster et al. 1989; Zabik et al. 1995). Thus an exposure model that assumes consumption of a fish filet with the skin on will lead to higher exposure estimates than will a consumption model assumes skin-off filets. Although we may be able to accurately model the bioaccumulation of contaminants into aquatic organisms, we must further be able to understand the partitioning within the organism to adequately understand the potential for human exposure and adverse human health outcomes.

A further difficulty in estimating the concentration of contaminants in fish is the fact that we often conservatively assume that an organism of interest spends all of its life in proximity to the area containing contaminated sediments. Thus the modeled concentration of contaminants in migratory species at the top of the food chain may he higher than actually observed because of the fact that these species spend only a fraction of time at the contaminated site. There are current models that attempt to account for this phenomenon and adjust risk estimates accordingly (Linkov et al. 2002; Von Stackelberg et al. 2002). These models result in lower risk estimates associated with a given level of contaminants, leading to higher sediment cleanup levels. A practical difficulty in applying these models is that the cost of collecting sufficient data to adjust for migration patterns (such as fish tagging studies) may be so high as to offset any cost savings from a reduced amount of sediments requiring remediation. Furthermore, assuming that conditions at the "other locations" are pristine, while allowing higher site-specific cleanup goals, may slowly raise the regional baseline concentration of contaminants to potentially undesirable levels.

In summary, human health can be used as an endpoint of concern in assessing risks that result from contaminated sediments. Using our knowledge of the bioaccumulation of sediment contaminants and subsequent biomagnification along a food chain, we can use the exposure–response models described in this section to estimate human health risks. Risk assessment models can be run in a forward direction to determine risks associated with current sediment contaminant levels. Alternatively, the models can be run in reverse, using an acceptable risk threshold to back calculate a sediment concentration protective of human health. However, our ability to precisely estimate human health risks is limited. Much of the variance in our ability to estimate these risks is not due to model or parameter uncertainty, but rather to natural variability in human exposure and susceptibility to these contaminants. We could have perfect models of bioaccumulation and still have highly variable risk estimates. A danger in running a risk assessment model in reverse is that if these uncertainties are propagated, either through selection of conservative point estimates or by using Monte Carlo simulations, there are likely to be allowable concentrations of contaminants that are either at or below naturally occurring or regional diffuse background levels for inorganic and organic contaminants respectively. Because much of this variance is due to natural variability, only an extraordinary amount of research can improve our ability to precisely estimate human health risks. The goal, therefore, is to adequately characterize this vari-

ability and to use this knowledge to make management decisions protective of human health.

Summary

Existing effects-based SQG approaches were not designed or intended to be protective of effects through bioaccumulation, either by interpreting risk of bioaccumulated contaminants or through food web transfer. While it is possible to develop SQGs protective of bioaccumulative effects, a number of issues must be considered to reduce uncertainty and ensure that the resulting guidelines are meaningful. One issue relates to potential discrepancies between laboratory and field-collected data. In general, laboratory-based bioaccumulation studies provide information that is representative of tissue residues obtained from field-collected organisms. However, differences in route and duration of exposure, seasonality, lipid content, etc. can lead to differences and reduce the reliability of laboratory to field comparisons. Appropriate consideration of such factors can be used to reduce uncertainty and improve the predictive ability of laboratory estimates. While other tools exist for estimating bioaccumulative potential of sediment-associated contaminants such as TBP modeling for nonionic organics, the use of biomimetic devices (e.g., SPMDs and SPMEs), or specialized extraction techniques (e.g., gut juice), these currently result in estimates with higher degrees of uncertainty than either laboratory- or field-based exposures. Another important issue is interpreting the significance of measured tissue residues. Understanding the potential consequences of bioaccumulation requires linking measured tissue residue levels with associated effects. While a number of databases summarize available residue effect information, there is a general paucity of available data, which limit the usefulness of this approach. One final issue relates to the reliability of risk-based approaches for establishing the potential for effects in higher trophic levels. Currently, the best estimates of bioaccumulation potential and resulting effects are for organisms in direct contact with the sediment. Generally, the further removed from direct exposure (i.e., higher in the food chain), the greater the uncertainty in the estimate of bioaccumulation and hence the greater the uncertainty in the estimate of risk. To ensure environmental protection of higher trophic levels (including humans), often overly conservative assumptions are used. Accuracy of these risk based estimates can be improved with development and application of region or site-specific data relating to food web structure (including area use factors) and trophic transfer coefficients.

Currently, there appear to be 2 potential types of SQGs that could be developed for assessing potential effects of contaminant bioaccumulation from sediments: 1) direct guidelines based on tissue residue effects data and 2) guidelines that incorporate the indirect effects through trophic transfer of contaminants. A primary concern in developing any meaningful guideline is addressing uncertainty. While it is possible to develop site-specific SQG values protective of effects through bioaccumulation, any approach should be adequately validated with field-collected data prior to implementation.

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